ULTRASONOGRAPGHY – A DIAGNOSTIC AID FOR CARPAL TUNNEL SYNDROME

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CERTIFICATE

This is to certify that the materials for this thesis entitled **"ULTRASONOGRRAPHY – A DIAGNOSTIC AID FOR CARPAL TUNNEL SYNDROME"** has been taken from the patients referred to the Dept. of Physical Medicine and Rehabilitation for nerve conduction studies, Christian Medical College and Hospital, Vellore.

Also certified that this thesis is the bonafide work of Dr. Bobeena Rachel Chandy, Post Graduate Registrar of this Department, under my guidance and supervision.

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CONTENTS **PAGE NUMBER**

INTRODUCTION

Carpal tunnel syndrome is a common clinical condition which is defined as a spectrum of signs and symptoms involving the hand and wrist due to the compression of the Median nerve as it passes through the carpal tunnel. Although the condition was first noted in medical literature in the early 19th century, the first use of the term “carpal tunnel syndrome” was in 1938(1).The condition was identified by orthopedician, Dr. George S. Phalen, of the Cleveland Clinic Foundation, as a painful disorder that afflicts workers whose jobs include repetitive wrist movements in the 1940s. He also described the clinical sign “Phalen’s manoeuvre”, which is named after him, for carpal tunnel syndrome, in 1948, at a meeting of the American Surgery for the Hand(1).

The history of carpal tunnel goes way back to 1854, when Sir James Paget, a British surgeon and pathologist, first reported median nerve compression at the wrist following a distal radius fracture. His first patient was a man who developed pain and impaired sensation in the hand from the trauma of a cord drawn tightly around his wrist. In a second case, tardy median nerve palsy was a consequence of a distal radius fracture; this patient improved with wrist immobilization and thus was also the first description of treatment with a neutral wrist splint, a method still in use today(2). Three decades later, James Putnam(3) (1880) presented a clinical series of 37 patients with the skin, giving rise to what is popularly known as numbness recurring periodically, coming on especially at night, “….in some cases simply letting the arm hang out of the bed or shaking it about for some moments would drive the numbness way.”(3)

A landmark article by Marie and Foix (4) (1913) provided the first comprehensive clinical and pathological assessment of a non traumatic median nerve lesion at the wrist. They reported to the French Neurological Society a case of an 80–year–old man with

bilateral thenar eminence atrophy. An important comment in the same article stating that, ". . . transection of the ligament could stop the development of these phenomena"(4) was neglected for decades.

The most common treatment option in the first 4 decades of the 20th century was a cervical rib excision as it was thought that CTS was caused secondary to proximal nerve compression. Influential physicians of the times, W. W. Keen(5) (1907), Kinnier Wilson(6) (1913), and Sargent(7) (1921) popularized this approach, concluding that a cervical rib, in patients with aberrant innervation of the opponens pollicis and abductor pollicis brevis by the seventh cervical root, could compress the C7 root and produce thenar atrophy and sensory changes in the first 3 fingers. It was the clinical failure of the cervical rib resections that led to a fundamental reconsideration of the pathophysiology of CTS. The first recorded carpal tunnel release surgery was performed by H. Galloway(8) in 1924. In a 1925, he described a woman with clinical signs and symptoms of CTS who experienced improved sensation of her index finger following surgical exploration of the median nerve at the wrist. Unfortunately, this was followed by the development of a neuroma and the reoccurrence of symptoms. Nine years later James Learmonth (9) (1933) described a detailed case report of the surgical transection of the transverse carpal ligament.

In the 1950s, Phalen and colleagues(10-12) published a series of landmark articles solidifying and further defining CTS as a clinical syndrome resulting from median nerve compression at the wrist. He confirmed the usefulness of Tinel's sign as the quintessential manoeuvre to provoke sensory phenomena in CTS(12): Tinel's sign over the median nerve at the wrist was elicited in every case. This is a tingling sensation, radiating out into the hand, which is obtained by light percussion over the median nerve at the wrist. He also described an additional provocative test now known as the Phalen's

sign: Although pressure within the carpal tunnel is increased by extension of the wrist, the numbness and paresthesia in the fingers of these patients could be increased by sharply flexing the wrist for a period of 60 seconds. Prompt improvement in these symptoms would result with release of the wrist from the flexed position(12). Finally, he concluded that transection of the carpal ligament could decompress the nerve sufficiently to restore its normal function(10).

It took more than 4 decades from the initial recognition that CTS was caused by median nerve compression at the wrist until transaction of the transverse carpal ligament became the main therapeutic option for CTS.

CTS became widely known among the general public in the 1990s because of the rapid expansion of office jobs with an estimated lifetime risk of 10% and an annual incidence of 0.1% among adults. Its prevalence is 5% in the general population with a female preponderance ranging from 3:1 to 23:1(13, 14). CTS displays a constellation of symptoms and signs in the hand and sometimes also in the upper limb. Diagnosis is mainly based on the history of symptoms (pain, numbness, tingling and burning sensation in the hand), provocative factors (sleep, repetitive movement of the wrist), mitigating factors (shaking the hand, changes in hand posture). The clinical presentation usually is of pain in the wrist and hand, paresthesia and weakness, which may give rise to the suspicion of carpal tunnel syndrome. However these symptoms may mimic other conditions like cervical radiculopathy, polyneuropathy or proximal Median nerve entrapment. Hence identifying the cause for the symptoms and the presence of any secondary etiological factors is made by Electrodiagnostic tests which are considered as the gold standard.

Neurophysiological diagnosis of carpal tunnel syndrome was first established by Simpson in 1956 by demonstrating focal slowing of median nerve conduction at the wrist(15).

Nerve conduction studies are highly specific (16) but may not be diagnostic in 10-25% of patients with clinical evidence of CTS depending on the severity of disease and the type of nerve conduction technique used (16, 17)(Duncan et.al.,1999; Jablecki et.al., 2002). It has a substantial rate of false positives in normal, asymptomatic individuals(13) (Atroshi et.al.). Despite the drawbacks of NCS, it is considered as the gold standard for diagnosis of CTS.

With recent advances in computer technology and equipment miniaturization, the clinical application of diagnostic Ultrasonography has spread across various medical specialties. Diagnostic Ultrasonography is popular in terms of its non-invasiveness, lack of radiation, readiness of use, cost-effectiveness and its ability to make dynamic examinations possible.

Peripheral nerve Ultrasonography is emerging as a promising diagnostic tool for entrapment neuropathies particularly CTS. This can be diagnosed by demonstrating an increase in the cross sectional area of the media nerve in the carpal tunnel (18-33). Ultrasonography provides a simple non-invasive means of visualizing peripheral nerve pathology. In the recent years MRI imaging has also become an alternative but the cost of the investigation makes it a poor alternative diagnostic tool.

This study is performed to assess the diagnostic role of Ultrasonography in CTS and it co-relation with the present day gold standard of NCS.

AIMS AND OBJECTIVES

1. To determine the cross-sectional area of the Median nerve in patients with symptomatic wrists electrophysiologically diagnosed for carpal tunnel syndrome, as compared to normal individuals.

2. To assess the specificity and sensitivity of Ultrasonography as a diagnostic tool for the diagnosis of Carpal tunnel syndrome and compare it to electrophysiological studies (gold standard for diagnosis of carpal tunnel syndrome)

3. To determine the use of Ultrasonography as an alternate investigational modality for diagnosis of Carpal tunnel syndrome.

REVIEW OF LITERATURE

Distal median neuropathy is the most common entrapment neuropathy affecting the upper extremity, the usual site of compression occurring in the wrist as the nerve passes under the flexor retinaculum in the carpal tunnel. Carpal tunnel syndrome is a common diagnosis with an estimated lifetime risk of 10% and an annual incidence of 0.1% among adults(34, 36). These estimates are undoubtedly conservative because they are based on data collected prior to the substantial increase in work-related cases of CTS in the 1980s and early 1990s and the concomitant increased awareness of this condition(37, 38). It is one of the most widely recognised occupational health conditions; particularly in industries where work involves high force/pressure and the repetitive use of vibrating tools. Also with the rapid expansion of office jobs and with the wide use of computers, the incidence of CTS has gone up. More recent estimates of the prevalence of CTS in the general population are 0.6% in men and 5.8% in women(39). Now, data from Sweden in which the prevalence of carpal tunnel syndrome in the general population reported suggests an overall prevalence of 2.1%(13).

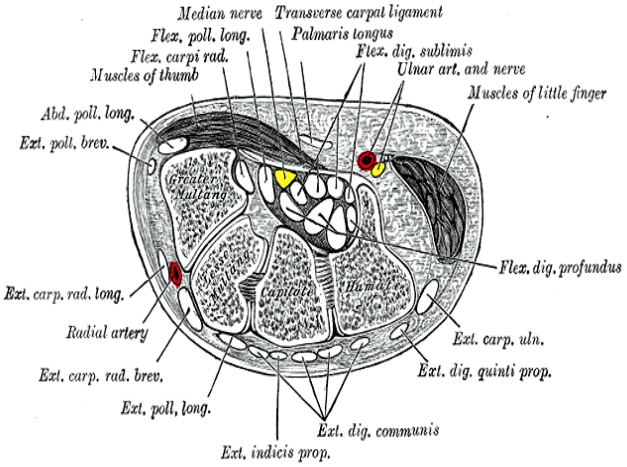
ANATOMY

The Median nerve is derived from the brachial plexus with a contribution from both the lateral and medial cord. The lateral cord is the larger of the two and is composed of fibres from the spinal levels C5, C6 and C7, while the medial cord consists of fibres from C8-T1. The lateral cord contributes mainly to the sensation of the thenar eminence, thumb, index and middle fingers as well as motor fibres to the proximal median forearm muscles. The medial cord supplies majority of the motor fibres of the distal median muscles of the forearm and the hand as well as sensory fibres to the lateral half of the ring finger.

The median nerve descends in the upper arm giving off no motor or sensory branches. In the antecubital fossa, the nerve runs adjacent to the brachial artery. Passing into the forearm, the nerve runs between the two heads of the pronator teres before innervating the Flexor carpi radialis, flexor digitorum sublimis and the palmaris longus. The largest branch of the median nerve, the anterior interosseus nerve, is then given off next in the proximal forearm, inervating the flexor pollicis longus, the medial head of the flexor digitorum profundus to digits 2 and 3 , and pronater quadratus muscles.

Just proximal to the wrist and carpal tunnel, the palmer cutaneous sensory branch arises, running subcutaneously to supply sensations over the thenar eminence. The median nerve then enters the wrist through the carpal tunnel which is formed by the carpal bones on the floor and the thick transverse carpal ligament forming the roof(40). The inlet of the carpal tunnel is taken as the proximal margin of the flexor retinaculum between the scaphoid tubercle and the pisiform bone and the tunnel outlet is the distal margin of the flexor retinaculum between the trapezium and the hook of the hamate(33)

Figure 1: Cross section across wrist.



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In addition to the median nerve, nine flexor tendons to the digits and thumb traverse the carpal tunnel. In the palm, the median nerve terminates into motor and sensory divisions. The motor division supplies lumbrical 1 and 2, and gives off the recurrent thenar motor branch which supplies the muscles of the thenar eminence that is opponens pollicis, flexor pollicis brevis and the abductor pollicis brevis. The sensory fibres of the median nerve that pass through the carpal tunnel supply sensations to sensations to the thumb, index, middle and lateral half of the ring finger. Hence in compression of the nerve in the carpal tunnel, the sensations of the thenar eminence are not affected on examination, but that of the thumb, index, middle or lateral half of the ring fingers (40).

AETIOLOGY

The aetiology of CTS is multifactorial, with local and systemic factors contributing to varying degrees. There are two distinct varieties of carpal tunnel syndrome – acute and chronic. The acute compression of the median nerve occurs infrequently and is due to rapid and sustained rise of pressure in the carpal tunnel leading to increasing and severe pain in the wrist with paresthesia and impaired sensations in the distribution of the median nerve(41). This is commonly associated with a fracture of the radius as described by Sir James Paget in 1854(42). It is also associated with burns, coagulopathy, local infections and injections. The chronic form is much more common and symptoms can persist for months to years. However, in only 50% of the cases is the cause identified. This can be divided into local, regional and systemic causes summarized in the following table:

Table 1 (43): Causes of carpal tunnel syndrome

A. LOCAL CAUSES
• Inflammatory: e.g. tenosynovitis, histoplasma fungal infection, hypertrophic synovium • Trauma: e.g. Colles' fracture, dislocation of one of the carpal bones • Tumours: e.g. Haemangioma, cyst, ganglion, lipoma, neuroma etc. • Anatomical anomalies: e.g. thickened transverse carpal ligament, bony abnormalities, abnormal Muscle bellies, persistent median artery.
B. REGIONAL CAUSES
• Osteoarthritis • Rheumatoid arthritis • Amyloidosis • Gout
C. SYSTEMIC CAUSES
• Diabetes • Obesity • Hypothyroidism • Pregnancy • Menopause • Systemic lupus erythematosus • Scleroderma • Dermatomyositis • Renal failure • Long-term haemodialysis • Acromegaly • Multiple myeloma • Sarcoidosis • Leukemia • Alcoholism • Haemophilia

Carpal tunnel syndrome is common in pregnant women(44-47). It is usually diagnosed in the third trimester of pregnancy and it is often bilateral. In majority of the patients

symptoms will resolve either spontaneously or will respond to conservative treatment after delivery(44, 48, 49).

ROLE OF OCCUPATION –

Carpal tunnel syndrome is the most common form of Repetitive Trauma disorder (RTD). In 1995, there were approximately 308,000 trauma related musculoskeletal disorders, representing nearly 62% of all occupational illness cases reported to the US Bureau of Labour Statistics(38). Brain *et al* were the first to implicate occupation as a causal factor in CTS(50). At risk occupations include, grinders, cashiers, and meat packers, workers sewing car seats, aircraft engineers, grocery store workers, and small part assembly liners. The physical factors implicated and extensively studied in relation to occupational CTS include repetition, force, posture, external pressure, and vibration. Repetition is the most widely recognized risk factor for occupational CTS. In epidemiological studies high repetition is defined either by the frequency of the task or the percentage of time spent on repetitive work. A high repetitive job is defined as one which involves the repetitive use of awkward wrist movements lasting less than 30s or when more than 50% of work time is spent performing tasks that involve repetitive awkward wrist movements(51). Experimental studies have shown a higher incidence of CTS in workers who are involved in high force and repetitive work compared to workers who are not(51-53). Silverstein *et al* examined the association between high force/repetitive movements and CTS among 652 workers from 39 jobs from seven different industrial areas(51). The authors noted a prevalence of 5.6% among workers in high force and high repetitive jobs compared to 0.6% among workers in low force and low repetitive jobs. The authors showed occupation to be a risk factor only when high force and high repetition are present, but the accuracy of their estimated ratio of 15.5 (95% confidence interval, 1.7-141.5) had the

draw back of small sample size. High repetitiveness seems to be a greater risk factor than high force but neither was statistically significant alone(51). In a case–control study, Armstrong & Chaffin compared patients with CTS to asymptomatic people amongst 18 sewing machine operators and noted that cases that used pinch grip (opposition of the thumb and the distal joints of four fingers) exerted more force than controls(54). The authors also noted that cases tended to use non-neutral postures more often and exerted more force in these postures(54). Several epidemiological studies have shown that force is an independent risk factor for CTS, but the dose–response relationship is not clear. In the study by Silverstein *et al (51)*force was a weaker risk factor than repetition, but in the report by Chiang *et al (55)* force was a stronger risk factor than repetition. Force and repetition increased the risk of CTS in a cumulative way in the Silverstein study. The odds ratio for high force and high repetition group was 15.5 in the Silverstein study. However, Chiang *et al* showed odds ratio for repetition was 1.1 and did not find a significant association between repetition and force(55).

Several studies examining carpal tunnel pressure (CTP) in healthy subjects indicate that the greatest increase in CTP occurs following wrist flexion and extension(56, 57). In an experimental study on 17 healthy volunteers, Rempel *et al* measured intra-compartmental pressures using a saline filled catheter introduced into the carpal tunnel(58). The authors noted highest mean intra- compartmental pressure (55mm Hg) during full supination and 90 degree metacarpophalangeal (MCP) joint flexion and lowest pressure at 45 degrees of pronation and 45 degrees of MCP joint flexion. The authors speculated that the increased carpal tunnel pressure at full supination and at 90 degree MCP flexion changes the orientation of the tendons, thereby, increasing the volume of the carpal tunnel predisposing to CTS. The authors suggested that the lowest pressures achieved by position should be considered in job and tool design(58).

In a review of 15 cross-sectional studies involving 32 occupational or exposure groups and six-case studies, Hagberg *et al* noted a high prevalence of CTS in occupations requiring high force and high repetitive manual movements(59). Occupational risk factors alone do not explain the occurrence of CTS and it is proposed that a combination of several factors is involved. The majority of CTS is attributable to patient related factors (intrinsic risk factors). Several studies have noted that the occurrence of CTS is correlated with unhealthy habits and lifestyle(60, 61). This was supported by an analysis that showed that 81.5% of the explainable variation in electro-physiologically defined CTS was attributable to body mass index, age, and wrist depth to width ratio, whereas only 8.29% was due to job related factors41. In a study comparing workers with and without CTS, Nathan and colleagues noted that there was a 19% greater lifetime use of tobacco, 75% greater history of alcohol abuse, and 5% greater use of caffeine in workers with CTS36. Furthermore, the authors reported that, current tobacco, caffeine, and alcohol consumption independently predicted 5% of the risk for CTS in female workers(62). Several studies have noted high incidence of CTS in patients with high body mass index (61, 63-65). Garland *et al* found that gender was a more predictive risk factor for CTS than exposure to high risk occupations(66). In a series of 654 hands with CTS, Phalen did not observe any relation between CTS and occupation. Furthermore, he argued that occupational trauma is seldom the precipitating factor in the production of CTS(1). It is important to establish the nature of risk factor and the interaction between intrinsic and extrinsic factors. In a longitudinal study of predictors of CTS in industrial workers over a period of 17 yrs, Nathan *et al* did not find an obvious relationship between the incidence of carpal tunnel syndrome and repetitive work. However, the authors noted high incidence of carpal tunnel syndrome in overweight people and in females(67). One of the major drawbacks of studies that show a positive association

between occupation and CTS is the wide variety of criteria used to diagnose CTS. Studies conducted in the 1980s depended on patients' self-reported symptoms and physical signs to establish the diagnosis of CTS. Ideally, the diagnosis of CTS should be based on combination of symptoms, physical signs and nerve conduction studies. Furthermore, studies relied upon patients to report the degree of occupational exposure. The most often sited publication linking the occupation exposure to high repetitive and force and the increased incidence of CTS relied on patient reported symptoms and physical examination for the diagnosis of CTS. Furthermore, the authors also did not define what constitutes high force and repetition(51). Despite the use of much more rigorous methods to establish the diagnosis of CTS, conflicting results were published in the 1990s linking occupation and CTS. Stetson *et al* examined workers from several industries and noted significantly lower sensory amplitudes and longer motor and sensory latencies on nerve conduction studies in occupations involving high repetition and force(68). Osorio *et al* studied 56 grocery store workers and found strong associations between forceful and repetitive wrist movements and the prevalence of CTS (69). However, several other studies did not find any substantial evidence linking specific occupations and the prevalence or severity of impaired sensory conduction of the median nerve at the carpal tunnel(62, 70). However, the most recent systematic literature review on the role of occupation in carpal tunnel syndrome by Palmer *et al*, found that the regular use of hand-held vibrating tools increased the risk of CTS by more than 2-fold (71). The authors also found substantial evidence for high risk of CTS in occupations requiring high repetitive flexion and extension at wrist and also forceful grip(71). However, the authors did not find evidence between the work on keyboard and computers and CTS.

PATHOPHYSIOLOGY

The exact pathogenesis of CTS is not clear. Several theories have been put forward to explain the symptoms and impaired nerve conduction studies. The most popular ones are mechanical compression, micro-vascular insufficiency, and vibration theories.

According to mechanical compression theory, symptoms of CTS are due to compression of the median nerve in the carpal tunnel. The major drawback of this theory is that it explains the consequences of compression of the nerve but does not explain the underlying aetiology of mechanical compression. Brain and colleagues attributed the symptoms of CTS to spontaneous median nerve compression in the carpal tunnel(50). The term 'spontaneous' was used due to lack of clear association between wrist joint deformities and symptoms. The compression was believed to be mediated by several factors such as exertion strain, overuse, repeated or prolonged wrist extension, prolonged grasping of tools, and unaccustomed manual work(50).

The micro-vascular insufficiency theory proposes that the lack of blood supply leads to depletion of nutrients and oxygen to the nerve causing it to slowly lose its ability to transmit nerve impulses. Scar and fibrous tissue eventually develop within the nerve. Depending on the severity of injury, changes in the nerve and muscles may be permanent. The characteristic symptoms of CTS, particularly tingling, numbness and acute pain, along with acute and reversible loss nerve conduction are thought to be secondary to ischemia of the affected nerve segment(43). Seiler *et al* showed (by laser Doppler flowmetry) how normal pulsatile blood flow within the median nerve was restored within 1 min of transverse carpal ligament release. The authors concluded that ischemia likely plays a significant role in the aetiology of CTS(72). A number of experimental studies support the theory of ischemia due to externally applied compression and due to increased pressure in the carpal tunnel(52). The development of

ischemia and therefore, symptoms, will vary according to the integrity of the blood supply of the nerve and the systolic blood pressure. Kiernan *et al* found that the conduction slowing in the median nerve can be explained by ischemic compression alone and may not always be attributable to disturbed myelination(73). Tucci *et al* noted five times higher levels of interleukin-6, maloionaldehyde bis- (diethyl acetal) and prostaglandin E2 at the time of surgery in patients with CTS compared to asymptomatic volunteers(74). The authors concluded that such alteration may be the result of oxidative changes following repetitive ischemia and reperfusion injury.

According to the vibration theory the symptoms of CTS could be due to the effects of long-term use of vibrating tools on the median nerve in the carpal tunnel(52). Lundborg *et al* noted epineural oedema in the median nerve within days following exposure to vibrating hand-held tools. Furthermore, the authors also noted similar change following mechanical, ischemic, and chemical trauma. Interestingly, the authors also report animal studies that show a temporary accumulation of smooth axoplasmic structures and deranged axoplasmic structures following a short exposure to a vibrating force(75).These changes were first noted in unmyelinated fibres that serve sympathetic activity; a loss of which could reduce micro-vascular flow to the median nerve leading to disruption of its myelin sheath and decreased motor conduction velocity(75). One of the hallmarks of chronic compressive neuropathies is demyelination(76).Damage to the myelin sheath of the median nerve impairs saltatory impulse transmission and nerve conduction|(77). Extensive demyelination and persistent compression eventually result in direct axonal damage with onset of Wallerian degeneration distal to the site of injury. These changes are more likely to occur in elderly patients with reduced regenerative capacity of the neurons and the Schwann cells(78).

CLINICAL FEATURES

The symptoms vary depending upon the severity of the disease. In early stages, patients usually complain of symptoms due to the involvement of the sensory component of the median nerve and only later report symptoms from involvement of motor fibres. Numbness is the cardinal sign of carpal tunnel syndrome. It is often bilateral and affects the radial three digits: but most often the index and middle fingers. At the beginning it may be intermittent but gets permanent as the median nerve damage progresses. The symptoms are almost always exacerbated during the early hours of the morning (brachialgia paraesthetica nocturna) due to the "foetal" sleeping position with wrist flexion but also because of altered fluid distribution in the lying position and increased blood flow to the limb. The symptoms are alleviated by shaking the affected hand (Flick sign) and can be provoked by prolonged wrist flexion as in driving(79). Symptoms of nocturnal paresthesia are reported to be 51-96% sensitive and 27-68% specific(80-82). Pain can be burning or prickling nature and may affect the limb as far proximal as the shoulder(83). Patients often present with complains of clumsiness. It may be due to finger hypoaesthesia, thenar atrophy or loss of proprioception. In some patients shoulder pain may be the presenting symptom but they will never have any objective evidence of sensory changes above the wrist. In Kendall's series of 327 patients, 313 (95.7%) reported paresthesia; 118 (38%) reported nocturnal symptoms only, 178 (58%) reported symptoms during the day and night, but worse at night, and 17 (5%) reported symptoms during the day only(84). In Phalen's experience, the typical history was that of a gradual onset of numbness and paresthesia(1).

In 1993, a clinical study was conducted to evaluate the reproducibility, internal consistency, validity and responsiveness to clinical change of scales for the measurement of the severity of symptoms and functional status. A self administered questionnaire was

developed as a result of the study, for the assessment of severity of symptoms and functional status in patients who have carpal tunnel syndrome (85). The functional scale assessment was as given below (85) :

FUNCTIONAL STATUS SCALE

On a typical day during the past two weeks have hand and wrist symptoms caused you to have any difficulty doing the activities listed below? Please circle one number that best describes your ability to do the activity.

Activity	No Difficulty	Mild Difficulty	Moderate Difficulty	Severe Difficulty	Cannot Do at All Due to Hand or Wrist Symptoms
Writing	1	2	3	4	5
Buttoning of clothes	1	2	3	4	5
Holding a book while reading	1	2	3	4	5
Gripping of a telephone handle	1	2	3	4	5
Opening of jars	1	2	3	4	5
Household chores	1	2	3	4	5
Carrying of grocery bags	1	2	3	4	5
Bathing and dressing	1	2	3	4	5

Table 2: Functional status scale from the Boston questionnaire (85)

In the symptom severity scale 11 questions are asked and based on the symptoms during a typical 24 hour period, the right answer from a choice of 5 is to be given

1. How severe is the hand or wrist pain that you have at night? 2. How often did hand or wrist pain wake you up during a typical night in the past two weeks? 3. Do you typically have pain in your hand or wrist during the daytime? 4. How often do you have hand or wrist pain during the daytime? 5. How long, on average does an episode of pain last during the daytime? 6. Do you have numbness (loss of sensation) in your hand? 7. Do you have weakness in your hand or wrist? 8. Do you have tingling sensations in your hand? 9. How severe is numbness or tingling at night? 10. How often did numbness or tingling wake you up during a typical night in the past two weeks? 11. Do you have difficulty with grasping and use of small objects such as keys or pens? (85).

In a systematic review of 10 studies which examined the psychometric properties of BCTQ, Leite *et al* concluded that BCTQ is highly reliable, responsive, and should replace any other non-standardized methods of assessment(86).

SIGNS

Several tests have been described which help in the diagnosis of CTS. None of these tests are diagnostic on their own. Most of the tests are complementary to each other rather than diagnostic of CTS. A combination of symptoms, signs and diagnostic tests should be taken into account when the diagnosis of CTS is made. The presence or absence of Characteristic physical findings have limited diagnostic value. The various tests are Tinel's sign, Phalen's sign, square wrist sign, closed fist sign, flick sign, Katz hand diagram, flexion and extension of wrist test, pressure provocation test, and tourniquet test. There are limited studies that have evaluated the diagnostic use of square wrist sign, flick sign, closed fist sign, and tourniquet test and hence these tests are not discussed in detail in this article. However, it is sufficient to say that prior to routine use of these tests; further evidence is required to support their effectiveness in the diagnosis of CTS. Diminished pinprick sensation (hypoalgesia) in the distribution of median nerve compared to the pinprick sensation over the ipsilateral little finger is a very useful diagnostic test in patients with CTS than abnormalities of other sensory modalities.

Tinel's sign

In this test, the examiner taps lightly over the site of the median nerve at the distal wrist crease. Development of tingling or discomfort in the fingers supplied by the median nerve constitutes a positive sign. Tinel described this sign in 1915 (87). He noted that a tingling sensation occurred when an injured nerve was percussed over its proximal stump

and speculated that this was a sign of axonal degeneration and intended his sign to be used in patients after blunt traumatic injury to follow the course of the regenerating nerve(88, 89). Tinel's sign is not a precise test and several factors can influence the outcome of the test. Firstly, its efficacy is reduced, as patients with CTS will have continually regenerating nerves at the distal wrist crease. The other limiting factor is the amount of pressure used to elicit the sign. Testing technique is important when the physician is eliciting Tinel's sign, and subtle differences in test performance probably account for some of the discrepancies in reported prevalence. It is difficult to quantify precisely how much pressure should be used to elicit the sign. The use of too much force or a sharp blow over a normal median nerve will produce finger tingling. This must not be interpreted as the presence of Tinel's sign. The Tinel's sign is associated with sensitivities of 23% to 67%, and specificities of 55% to 100% (81, 82, 88-92). In a review, Kuschner *et al* summarised the frequency of Tinel's sign and reported that it is positive from 8% to 100% of CTS patients (89). Tinel's sign is the least accurate test according to Mondelli *et al,* who did not find a combination of signs more useful than a single sign alone(92).

Phalen's test

Phalen and Kendrick described this test in 1957(1). Flexion of the wrist causes compression of the nerve between the transverse carpal ligament (TCL) and flexor tendons in the carpal tunnel, causing paresthesia in the median nerve distribution(91, 93) reproducing the patient's symptoms. Phalen performed the test by having the patient hold the forearm vertically with the elbows resting on the table and then allowing both hands to drop with complete wrist flexion for approximately one minute. The test is considered positive when paresthesia develops in less than one minute. Patients with advanced CTS

often note paresthesia in less than 20 seconds. The reported sensitivity ranges between 10% and 91% and specificity between 33% and 100%(81, 82, 90, 94, 95).

Katz hand diagram

This is a self–administered diagram, which depicts both the dorsal and palmar aspect of the patient's hands and arms. Patients use this diagram to mark the specific location of their symptoms, characterising them as pain, numbness or tingling, or other. The diagnosis is graded as classic, probable, possible or unlikely to be CTS based on criteria that appear in the hand diagram(81, 96). In diagrams classified as classic or probable the sensitivity of the test is 80% and the specificity is 90% for the diagnosis of CTS(96, 97). Katz himself reported a sensitivity of 64% and a specificity of 73%(96)

Square wrist sign

Kuhlman *et al* reported that a square-shaped wrist, where the anterior-posterior dimension of the wrist (at the distal wrist crease) divided by the medio-lateral dimension is greater than 0.7065,(98) and weakness of the abductor pollicis brevis were the two most sensitive signs (69 and 66% respectively). This test is associated with a sensitivity of 47% to 69% and specificity of 73% to 83% (88, 98).

The tethered median nerve stress test

LaBan described this test in 1986. It is performed by hyperextending the supinated wrist and the distal interphalangeal joint of the index finger for a minute. Patients with chronic carpal tunnel syndrome experience pain on the volar aspect of proximal forearm(99). LaBan noted that hyper extension of index finger causes distal excursion of the median nerve more than hyperextension of the adjacent fingers(100). Raudino evaluated this test

in 140 patients with electro- physiologically confirmed CTS and noted that the test was positive in 60 hands (42.8%) compared to the 56.4% positive rate with Phalen's sign and 42% positive rate with the Tinel's sign(101).

Pressure provocation test

A positive result in this test is the presence of pain, tingling and numbness in the distribution of the median nerve when the examiner presses with his/her thumb on the palmer aspect of the patient's wrist at the level of the carpal tunnel for 60 seconds. The test is seldom positive. The reported sensitivity is between 28% and 63% and specificity is between 33% and 74% (82, 88, 93).

Tourniquet test

A positive result is the development of paresthesia in the distribution of the median nerve when a blood pressure cuff around the patient's arm is inflated to above systolic pressure for a minute or two. The irritated and compressed median nerve is thought to be more susceptible to ischemia than the normal median nerve. However, even normal individuals can also develop the same symptoms and it is difficult to evaluate, especially in mild cases of CTS. The tourniquet's test sensitivity lies between 21% and 52% with a specificity between 36% and 87%(82, 90).

MOTOR EXAMINATION

Thenar atrophy is a late sign and signifies significant functional loss. Finger weakness associated with an inability to pinch or frequent dropping of grasped objects follows involvement of the motor component. Long-term involvement leads to thenar muscle atrophy with associated loss of thumb abduction and opposition strength. Diminished

sensation to pinprick in the median nerve distribution always precedes thenar atrophy. Thenar atrophy is seldom noticed by patients and may not be obvious even to the examiner when examined by looking down onto the palm. However, it will be readily appreciated by comparing both palms together (1)**.** In Phalen's series the atrophy of abductor pollicis brevis, opponens pollicis and flexor pollicis brevis was noted in 41% of hands(1). Abductor pollicis brevis is the most commonly affected muscle and testing its function with the "Pen test" is useful in making the diagnosis of CTS.

The role of Nerve Conduction Studies

The nerve conduction studies (NCS) measure the sensory and motor nerve conduction velocity in the median nerve at the level of the wrist. Sensory nerve conduction studies as opposed to motor conduction techniques are more likely to reveal an abnormality of the median nerve action potential propagation because the sensory fibres are usually affected first and to a greater degree than motor fibres. The antidromic sensory potentials are recorded by stimulation of the nerve at a proximal point and the nerve action potential is recoded distally. Although routine motor nerve conduction studies to the abductor pollicis brevis (APB) are less sensitive than sensory nerve action potentials (SNAPs), it is still important to perform them in order to assess the extent of the pathologic involvement with respect to motor fibres. The distal motor latency to APB is the primary parameter examined by most practitioners. A distal motor latency delay in excess of that expected from normals suggests pathological involvement of motor fibres in the carpal tunnel(102, 103). The motor nerve conduction velocity from elbow to wrist is measured using surface electrodes. The needle electromyographic examination is less sensitive than nerve conduction studies in general and sensory studies in particular with respect to detecting early CTS. This is primarily because the main pathologic neural alteration in

early CTS is demyelination causing slowing of sensory action potential propagation across the carpal tunnel region with prolongation of the SNAP latency. Axonal loss of sensory fibres is minimal in mild CTS resulting in SNAP amplitude reduction that may be missed because of the wide range of normal amplitudes. If motor axonal loss is slowly progressive, the denervated muscle fibres can be reinnervated by the neighbouring intact axons through the process of collateral sprouting. In this way, there maybe slow formation of denervated muscle fibres that are reinnervated relatively rapidly thus minimizing the total number of orphaned muscle fibres at any time(104).

According to the summary statement put forth by the American Association of Electrodiagnostic Medicine, American Academy of Neurology and American Academy of physical Medicine and Rehabilitation in patients with suspected CTS, the following Electrodiagnostic studies are recommended: 1. Perform a median sensory NCS across the wrist with a conduction distance of 13 to 14 cm (Technique G). If the result is abnormal, comparison of the result of the median sensory NCS to the result of a sensory NCS of one other adjacent sensory nerve in the symptomatic limb (Standard). 2. If the initial median sensory NCS across the wrist has a conduction distance greater than 8 cm and the result is normal, one of the following additional studies is recommended: a. Comparison of median sensory or mixed nerve conduction across the wrist over a short (7 to 8 cm) conduction distance (Technique C) with ulnar sensory nerve conduction across the wrist over the same short (7 to 8 cm) conduction distance (Technique D) (Standard), or b. Comparison of median sensory conduction across the wrist with radial or ulnar sensory conduction across the wrist in the same limb (Techniques B and F) (Standard), or c. Comparison of median sensory or mixed nerve conduction through the carpal tunnel to sensory or mixed NCSs of proximal (forearm) or distal (digit) segments of the median nerve in the same limb (Technique A) (Standard). 3. Motor conduction study of the

median nerve recording from the thenar muscle (Technique H) and of one other nerve in the symptomatic limb to include measurement of distal latency (Guideline). 4. Supplementary NCS: Comparison of the median motor nerve distal latency (second lumbrical) to the ulnar motor nerve distal latency (second interossei) (Technique J), median motor terminal latency index (Technique I), median motor nerve conduction between wrist and palm (Technique E), median motor nerve compound muscle action potential (CMAP) wrist to palm amplitude ratio to detect conduction block, median sensory nerve action potential (SNAP) wrist to palm amplitude ratio to detect conduction block, short segment (1 cm) incremental median sensory nerve conduction across the carpal tunnel (Option). 5. Needle electromyography of a sample of muscles innervated by the C5 to T1 spinal roots, including a thenar muscle innervated by the median nerve of the symptomatic limb (Option)(16, 34, 105).

Table 3 (16): Comparison of pooled sensitivities and specificities of Electrodiagnostic techniques to diagnose carpal tunnel syndrome.

Technique	Pooled sensitivity*	Pooled specificity*
A. Median sensory and mixed nerve conduction: wrist and palm segment compared with forearm or digit segment	0.85† (0.83, 0.88)	0.98† (0.94, 1.00)
B. Comparison of median and ulnar sensory conduction between wrist and ring finger	0.85 (0.80, 0.90)	0.97 (0.91, 0.99)
C. Median sensory and mixed nerve conduction between wrist and palm	0.74† (0.71, 0.76)	0.97† (0.95, 0.99)
D. Comparison of median and ulnar mixed nerve conduction between wrist and palm	0.71 (0.65, 0.77)	0.97 (0.91, 0.99)
E. Median motor nerve conduction between wrist and palm	0.69† (0.64, 0.74)	0.98† (0.93, 0.99)
F. Comparison of median and radial sensory conduction between wrist and thumb	0.65 (0.60, 0.71)	0.99 (0.96, 1.00)
G. Median sensory nerve conduction between wrist and digit	0.65† (0.63, 0.67)	0.98† (0.97, 0.99)
H. Median motor nerve distal latency	0.63† (0.61, 0.65)	0.98† (0.96, 0.99)
I. Median motor nerve terminal latency index	0.62† (0.54, 0.70)	0.94† (0.87, 0.97)
J. Comparison of median motor nerve distal latency (second lumbrical) to the ulnar motor nerve distal latency (second interossei)	0.56‡ (0.46, 0.66)	0.98‡ (0.90, 1.00)
K. Sympathetic skin response	0.04 (0.00, 0.08)	0.52 (0.44, 0.61)

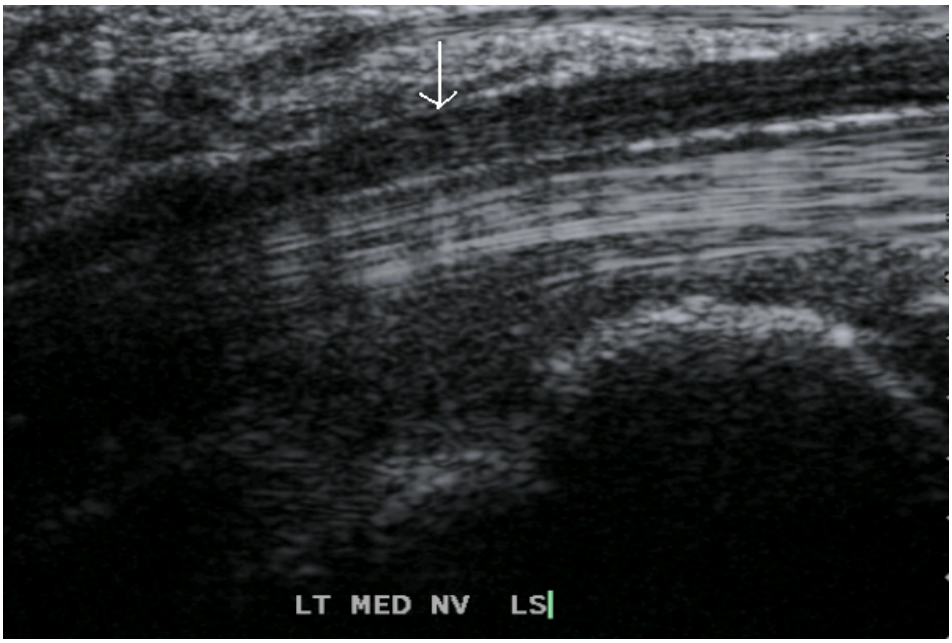
Median nerve conduction studies are the gold standard diagnostic tests with sensitivities between 49% and 84% and specificities of 95% and 99%(16, 34, 105). In entrapment neuropathies there will be a delay in the conduction velocity at the point of compression due to the demyelination of the nerve. In patients with clinical symptoms suggestive of CTS with normal sensory conduction velocity, measurement of both motor and sensory conduction velocity increases the diagnostic yield by 10%(106). Chang *et al* found that, in patients with normal sensory and motor conduction velocities, measuring the latency between the median and ulnar nerve for the ring finger and comparing it to the median and radial nerve latency for the thumb increases the diagnostic yield by another 10%(106). NCS not only allow a diagnosis of CTS but also help in the diagnosis of other

conditions presenting with similar symptoms e.g. cervical radiculopathy, polyneuropathy, other median nerve entrapment syndromes(94, 107). Although nerve conduction studies are the gold standard test for the confirmation of diagnosis of CTS, they have certain limitations. A small percentage of asymptomatic individuals can have positive NCS. Similarly, a small percentage of patients can have negative NCS despite symptoms suggestive of CTS. Atroshi *et al* randomly surveyed 2466 individuals in Sweden to find out the incidence of CTS in general population(13). 14.4% complained of pain, tingling and numbness in the distribution of the median nerve. However, only 4.9% of individuals with neuropathic symptoms had positive NCS. Furthermore, 18% of asymptomatic subjects had abnormal NCS9. In severe CTS cases, NCS results may not correlate with the clinical findings due to the varying nature of the impairment in different nerve fibres. In addition, nerve conduction studies will not accurately predict the recovery following release of the carpal tunnel, though neither do any of the other investigations predict this with any certainty (108). Therefore these studies suggest that NCS alone should not be used to diagnose, rather it should be based on presence of clinical symptoms, physical findings and positive nerve conduction studies taken together.

The role of imaging in the diagnosis of carpal tunnel syndrome

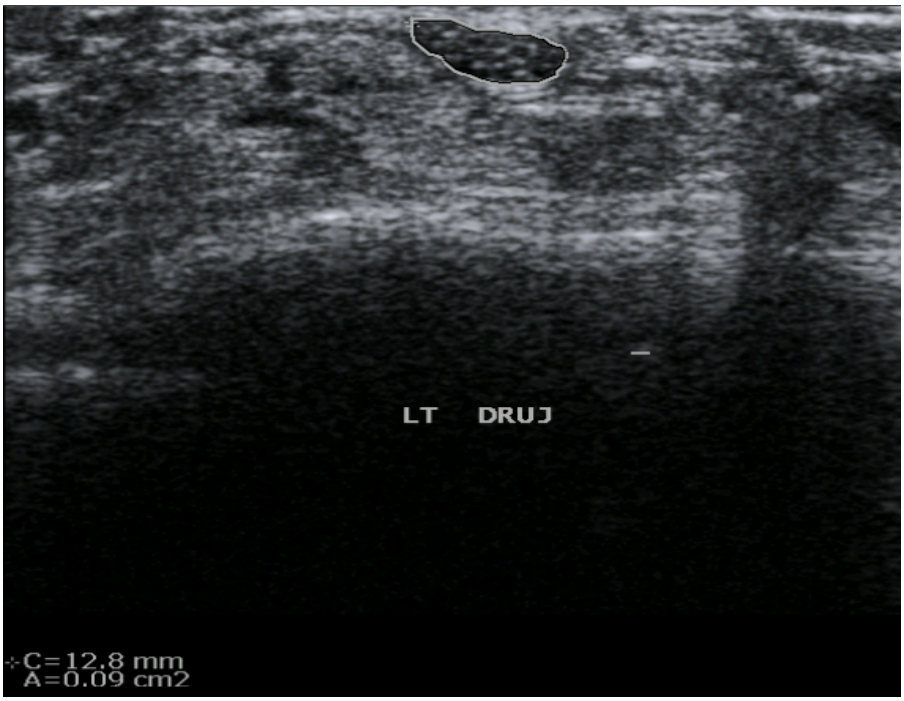
The sonographic anatomy of the median nerve, examined with a high-frequency probe, has a fascicular hypoechoic echotexture and is easily differentiated from tendons, which show a typical fibrillar hyperechoic pattern (Figure 2: longitudinal section of median nerve and adjacent tendon)

Figure 2: Longitudinal section of media nerve with adjacent tendon.



The median nerve is located between the flexor pollicis longus and flexor digitorum tendons, lying anterior to the latter. On transverse scans, the median nerve appears as an oval structure with long axis oriented parallel to the flexor retinaculum. The flexor retinaculum encloses the carpal tunnel anteriorly and is seen as a hyperechoic band separating the flexor tendons and the median nerve from the subcutaneous and cutaneous layers. Posterior to the tendons, the radial epiphysis and the carpal bones are seen as a hyperechoic line(109).

Figure 3: transverse image of median nerve at distal radio-ulnar joint.



The diagnosis of CTS is based mainly on clinical symptoms and signs and nerve conduction studies. There are many conditions associated with CTS, but in some patients, an underlying disease process cannot be identified and the description of "idiopathic CTS" is used. Some studies in patient without an attributable cause for CTS show that the anatomy of the hand, wrist and carpal tunnel may play a role for the development of CTS(110-112)encouraging the possible explanation of high prevalence of bilateral CTS. However, it was also reported that carpal canal size was not found to be a risk factor for CTS, implying other risk factors and work-related factors to be possibly responsible for the development of CTS(113). Any condition that exerts pressure on the median nerve at the wrist can cause this syndrome. Therefore imaging techniques are important to detect and exclude those possible pathologic causes. The biologic response of nerves to compression is a cascade comprising endoneural oedema, demyelination, inflammation, distal axonal degeneration, fibrosis, growth of new axons,

remyelination and thickening of the perineurium and endothelium(114). Sonography of patients with CTS finds the median nerve thickened. Endoneural oedema and thickening of the perineurium and endothelium are the probable causes of this finding. This seen on Sonography is an indirect finding of CTS and can be used as one of the diagnostic criteria.

Buchberger et al (31, 115) demonstrated the enlargement and deformation of the median nerve with Ultrasonography for the first time and described the characteristic features that could be found in patients with CTS. In a prospective study, Keles examined the role of US in 35 patients with a NCS confirmed diagnosis of CTS and compared it to 40 normal wrists. US measured the cross sectional area (CSA) of the median nerve, bowing of the flexor retinaculum (FR) and flattening of the flexor retinaculum(116). The CSA of the median nerve and bowing of the FR were significantly increased in patients with NCS positive CTS when compared to controls. The flattening of the FR had no correlation with diagnosis of CTS. In the work of Duncan et al (17), considered nerve flattening higher than 3.3, sensitivity was 38% and specificity 75%, while other authors with index higher than 3 found sensitivity 60% and specificity 76%(117). Therefore the index of nerve flattening and qualitative measurements such as longitudinal nerve entrapment showed low sensitivity and hence has less practical interest in the study of patients suspected of CTS.

Koyuncuoglu studied the role of US in 59 patients with clinical diagnosis of CTS with negative NCS findings by comparing their results with US findings in 30 normal wrists. They found a CSA of larger than 10.5mm in 18 patients compared to one wrist in the control group(118). El Miedany *et al* compared the results of US with NCS in a group of patients with CTS against a control group and observed a high degree of correlation between the US findings and NCS in diagnosing and in assessing the severity of CTS.

US also helped to identify the underlying cause of CTS and thus facilitated planning of treatment(119). Several studies evaluating sonographic accuracy for diagnosis of CTS found that ultrasound had a lower sensitivity (57-89%) but a higher specificity (47-94%) than Electrodiagnostic studies(17, 18, 28, 119-124). The sensitivity and specificity of sonographic features vary widely among published studies and the critical cut-off value for the nerve cross sectional area, at which nerve entrapment can be diagnosed, varies considerably from 9 to 15 mm2. The most consistent indicator seems to be the cross sectional area of the median nerve at the level of the pisiform bone and the hook of hamate, at the wrist crease(17, 28, 31, 115, 123, 125). The median nerve cross sectional area can be obtained by using either of the two method – direct or indirect. The direct technique is defined by tracing a continuous line around the inner hyperechoic rim of the median nerve with electronic callipers. The indirect technique is taken from the anteroposterior measurement (D1) and transverse distance (D2) of the inner median nerve calculated through the ellipsoid formula [pi(D1 X D2)/4](23).

Along with measuring the cross sectional area, flattening ratio, bowing of the flexor retinaculum, imaging can detect structural causes of nerve compression such as synovitis, ganglion, aberrant muscle and tumour as well as anatomic variations such as bifid median nerve and persistent median artery(126). Due to improved technology, there is better delineation of the perineurium, fascicles and septal structures within the nerve. This enables the examiner to detect atypical vascular structures within the carpal tunnel which maybe of importance before carpal tunnel release as is important to know the presence of a bifid nerve(126).

Cases of CTS associated with anatomic variations of the median nerve inside the carpal tunnel have been described(109, 126-128). These anatomic variants have also been described and classified into four groups by Lanz (129):

I: Variations of the course of the thenar branch

II: Accessory branches at the distal portion of the carpal tunnel

III: Divided or duplicated median nerve inside the carpal tunnel

IV: Accessory branches proximal to the carpal tunnel.

Group III of the Lanz classification is associated more frequently with CTS(129).

Due to improved technology, there is better delineation of the perineurium, fascicles and septal structures within the nerve. This enables the radiologist to differentiate high division proximally to the carpal ligament, with complete separation of fascicles and the perineurium, from a fascicle regrouping separated by septal structures but enveloped by a common perineurium. This latter variation though not described in anatomic sections, is referred to as a bifid nerve(126).

In the absence of knowledge of an anatomical variation, the persistent median artery may be injured leading to post operative risk of bleeding, haematoma formation, and fibrosis leading to recurrence of CTS. If the bifid nerve is not completely released, there will be persistence of the symptoms leading to re-exploration. It is therefore considered in recent times in the presence of a large number of studies conducted on the usefulness of Sonography that along with the clinical findings, Electrodiagnostic studies, help in aiding the diagnosis of CTS especially in providing better idea about the morphology of the nerve and its surrounding structures.

TREATMENT

There are several treatment options and they can be broadly categorised into surgical and non-surgical. Non-surgical methods are effective in patients with mild to moderate CTS. They are indicated in patients with no muscle weakness or atrophy, absent denervation (on electromyography needle examination), and with only a mild abnormality on nerve

conduction studies(130). Pregnant women with CTS rarely require surgical treatment. In the majority of patients symptoms will resolve either spontaneously or will respond to conservative treatment after delivery(46, 48, 49, 131). A Cochrane literature review (132) for randomized trials that compared surgical to non-surgical treatment of CTS included only one article(133). This study demonstrated significant clinical improvement in electromyography and symptoms reported at 1 year for surgical release over splinting with a cohort of 22 women. More recently, Gerritsen et al, published a second randomized study of surgical release versus splinting in 176 patients with moderate carpal tunnel syndrome, defined by clinical and electrophysiological testing (134, 135). Surgical patients had greater improvement in the number of nights waking up due to symptoms, and severity of symptoms, as well as on a general improvement scale. However, the evidence is less clear for patients with a shorter duration of symptoms or the use of conservative therapies other than splinting, such as physical therapy and ultrasound(135, 136). Although there is generally a lack of rigorous scientific support for non-surgical treatments for CTS (137), there is limited evidence of benefit for certain interventions. Common conservative treatments for CTS include wrist splints, hand therapy, non-steroidal anti-inflammatory drugs (NSAIDs), and corticosteroid injection into the carpal tunnel (138-142)[11-26]. Garfinkel (140) reported that yoga hand exercises resulted in improved Phalen sign compared with splints. Rozmaryn (141) reported that nerve and tendon gliding exercises coupled with traditional conservative care may reduce the need for surgery. Little evidence exists to support the use of NSAIDs for treating CTS. Celiker et al (138) found that corticosteroid injection into the carpal tunnel was superior to NSAID therapy and Davis and colleagues(139, 142) reported that ibuprofen combined with nocturnal splints did not improve outcomes more than chiropractic manipulation(140, 143, 144). Local injection of corticosteroid into the

carpal tunnel improves short-term clinical outcomes, as compared with oral corticosteroids, intramuscular corticosteroid injections, NSAIDs, or splints alone. (138, 145-147). Herskovitz and colleagues (148) demonstrated a short term improvement in global symptom scores for CTS with oral corticosteroid compared to placebo, but Chang could not find a dose response (149, 150). While these finding suggest some potential therapeutic benefits, none of these therapies either alone or in combination have been rigorously compared to surgery. Although not commonly used, Ebenbichler (151) found that focused ultrasound significantly improved symptom and electrophysiological outcomes compared with sham ultrasound. Vitamin B-6 has also been suggested as a treatment for CTS,(152) but two studies (36) failed to demonstrate improvement in outcome.

Surgical management

Surgery consists of division of transverse carpal ligament. This reduces the pressure on the median nerve by increasing the space in the carpal tunnel. Surgery is indicated in almost all patients with moderate to severe CTS. An absolute indication for CT release (CTR) is muscular atrophy(153). Two different types of surgical approaches are in use for the treatment of CTS; open and endoscopic release. Open CTR (OCTR) is the traditional option and still the recommended method of surgical treatment for idiopathic CTS. It was first performed by Herbert Galloway in 1924, though since then several modifications have been made to refine it (154). The classic OCTR uses a curved longitudinal inter-thenar incision, approximately 4 to 5 cm in length(155). It involves opening of subcutaneous tissue, superficial fascia and transverse carpal ligament and 2 to 3 cm of distal forearm fascia under direct vision. The canal is also inspected for mass lesions and anatomical abnormalities(43). Open carpal tunnel release is easy to perform and in majority of patients it leads to good symptomatic relief with a low complication

rate**.** In a series of 32 patients who underwent OCTR over a period of four years, 88% of patients reported

good functional and symptomatic improvement(156). The well recognised early complications are incomplete release of TCL, neuropraxia or injury to the median or ulnar nerve, inadvertent entry to Guyon's canal, injury to the palmer cutaneous or recurrent motor branch of the median nerve and injury to the superficial palmer arch or ulnar artery109. These complications are rare as surgery is performed under direct vision. The late complications are scar tenderness, loss of grip strength, pillar pain, and rarely reflex sympathetic dystrophy and bow stringing of flexor tendons. Pillar pain is a frequent complication of both open and endoscopic release procedures. The pillar pain is characterised by pain or tenderness in the thenar or hypothenar eminence. The incidence varies between 6 and 36%(157). It delays resumption of daily activities, return to work, and causes emotional distress all leading to an increased cost to health care system(158). The exact aetiology of pillar pain is not clear. However, the pain could be secondary to alteration of the carpal arch structures(159), oedema of the tissues superficial to TCL, injury to the cutaneous branches of the palm(160), or could be due to relaxation of the muscles of opposition and pinch following sectioning of TCL. As in other fields of surgery, less invasive techniques have been introduced into carpal tunnel surgery to facilitate earlier return to work and reduce post-operative pain and the first endoscopic carpal tunnel release was performed by Okutsu and his colleagues in Japan in 1987(161). Since its introduction several modifications of the technique have been described in the literature. There are several endoscopic approaches but the underlying principle is the same; to release transverse carpal ligament. ECTR techniques can be broadly divided into single portal and dual portal techniques depending on the number of ports used to access the carpal tunnel. The two most commonly used techniques are single-portal technique described by Agee(162) and two-

portal technique described by Chow(163). The reported success rates for surgical treatment range from 70 to 90%. In an extensive review of all articles on ECTR covering six different types of techniques, Jimenez *et al* found that the endoscopic release techniques offer similar success and complication rate as open surgery(164). The overall success rate for ECTR was 96.52% with a complication rate of 2.67% and a failure rate of 2.61%(164). The most common complications noted by the authors were paresthesia of the ulnar and median nerves, injury to superficial palmer arch, reflex sympathetic dystrophy, flexor tendons lacerations and incomplete division of TCL(164). The Cochrane database group reviewed all available evidence from randomized controlled trials comparing various surgical techniques in terms of efficacy in relieving symptoms, promoting early return to work and post-operative complications and found no strong evidence to favour alternative surgical techniques against the standard open technique. Specifically, they found conflicting evidence in support of endoscopic release leading to an earlier return to work and/or activities of daily living when compared to open CTR(165). These findings have been replicated by another meta-analysis study of randomized controlled trials comparing endoscopic and open carpal tunnel decompression which also found no conclusive evidence favouring ECTR with regard to symptom relief and return to work(166). However, they found that ECTR was associated with reduced scar tenderness and increase in pinch grip and pinch strength at 12 weeks follow up(166).

MATERIALS AND METHODS

The study was conducted in the Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore. The subjects were patients with symptoms of Carpal Tunnel Syndrome, referred from the out-patient services of the departments of Hand and Leprosy Reconstructive surgery (HLRS), Orthopaedics and PMR, between December 2006 and July 2008.

The total sample size of 100 subjects was required to find a hypothesized sensitivity of 90% with a 95% confidence interval of 85-95%. Of this number, 36 were in the patient group and 64 in the control group.

Definition of cases and data collection at initial evaluation:

The patient group were selected based on the diagnostic criteria put forward by the American Academy of Neurology (1993) (34) summarized here – paresthesia: pain; swelling; weakness or clumsiness of hand provoked or worsened by sleep; sustained hand or arm position; repetitive action of the hand or wrist that is mitigated by changing posture or by shaking of the hand; sensory deficit or atrophy of the Median nerve innervated thenar muscle; symptoms elicited by the Phalen's test (1 minute passive forced flexion of the wrist), performed on each patient.

A detailed clinical history and examination and neurophysiological evaluation were always performed. Laboratory investigations to diagnose any secondary cause for CTS were done for all patients. Only idiopathic CTS (with no aetiological factors) were included into the study.

The exclusion criteria included – 1. History of wrist surgery (including carpal tunnel injection) or fracture; 2. Clinical or electrophysiological evidence of an accompanying condition that mimics CTS or interferes with is evaluation, such as proximal Median

neuropathy, cervical radiculopathy or polyneuropathy; 3. History of underlying disorders associated with CTS such as diabetes mellitus, rheumatoid arthritis, pregnancy, acromegaly or hypothyroidism.

Control group:

64 healthy, age group matched subjects with no signs or symptoms of CTS were studied as the control group: 33 males and 31 females. The subjects in the control group were either healthy relative accompanying the patients during their visit to the hospital or from hospital staff. They were subjected to history and neurological examination to verify their normality. Nerve conduction studies and Ultrasonography of both wrists were done for all subjects included in the control group (total 128 wrists).

Patient group: The 36 patients who were included in the study were referred to PMR for nerve conduction studies. A detailed history was taken using the Boston Carpal tunnel questionnaire which assessed the symptoms (pain, paresthesia, numbness, weakness and nocturnal symptoms) and functional status (writing, buttoning, holding, gripping, opening jars, carrying grocery bags, household chores, bathing and dressing.). A neurological examination and laboratory investigations (blood sugars and thyroid functions) were also done to rule out any other cause for CTS. An informed consent was taken from all the patients.

Electrodiagnostic evaluation:

Electrodiagnostic studies were performed for all subjects included in the study according to the protocol put forth by the American Association of Electrodiagnostic Medicine recommendations. The machine used for the study was Medelec Synergy. The nerve

conduction study was in the same room and in similar temperature conditions. Standard tests for Median sensory and motor conductions were done looking at the distal latency, conduction velocity across the wrist and amplitude. The criteria for diagnosis of CTS were –

-Distal sensory latency recorded from the index finger (antidromic stimulation) using ring electrodes is >3.3 ms.

-Distal motor latency of Median nerve recorded from the abductor pollicis brevis and stimulating 3cms proximal to the distal crease of the wrist if exceeds 4.4 ms.

When standard tests mentioned above yielded normal results, a comparative median/ ulnar studies were done.

-Difference between distal motor latency of Median and Ulnar nerves > 1.1ms

-Difference between distal sensory latency of median and Ulnar nerves > 0.2ms. (15)

F wave was done for all patients.

In accordance to with the results of the Electrodiagnostic studies, the hands were categorized into 3 groups:

1. Mild to moderate CTS – characterized by slowing of the median sensory digit –wrist segment and normal distal motor latency or abnormal distal motor latency with or without delay in conduction velocity and diminished amplitudes.(35)
2. Severe CTS - absence of a median sensory response and abnormal distal motor latency with delayed conduction velocity and diminished amplitude or absence of motor conductions.(35).

Sonography:

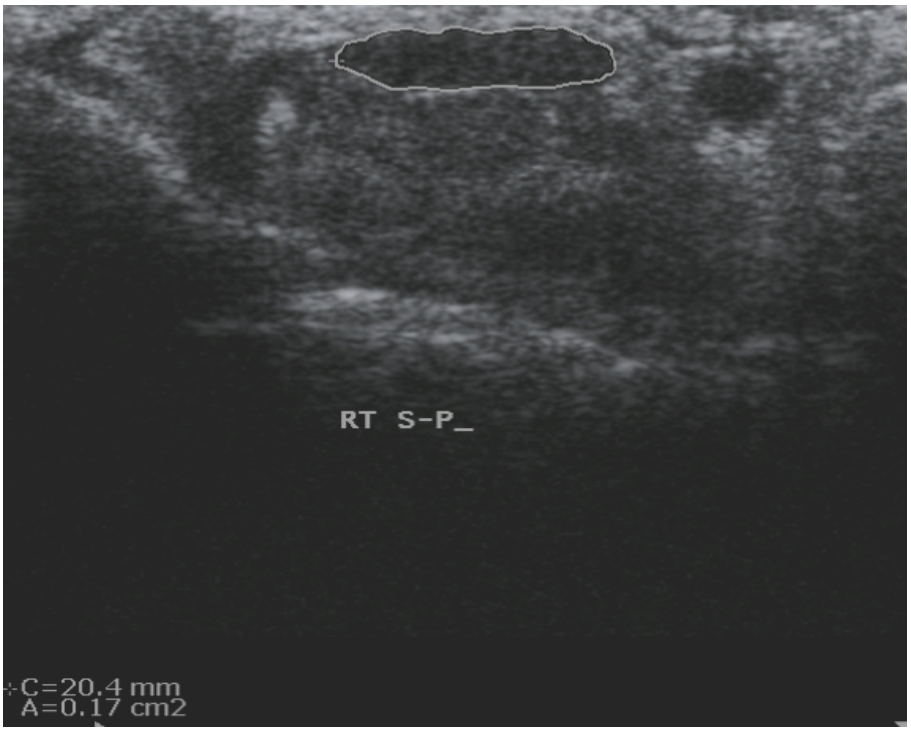
All 100 subjects underwent high resolution using Seimens Antares Ultrasonography machine with a 7-13 MHz linear array transducer. The radiologist was blinded to the Electrodiagnostic results. To ensure unbiased examination, the examiner was requested not to enquire about the symptoms and the patients were asked not to speak about the problem

during examination. The sonographic examination was done on the same day or within 3 days of the Electrodiagnostic study. The examination was performed with the patient seated in a comfortable position facing the radiologist with the forearm resting on the table in supination with the wrist in the neutral position and fingers semiflexed (fig.4, 5). Transverse images of the Median nerve were obtained at three levels: immediately proximal to the carpal tunnel inlet (distal radio-ulnar joint level), at the carpal tunnel inlet (level of scaphoid and pisiform) and at the carpal tunnel outlet (trapezium and hook of hammate level).

Figure 4

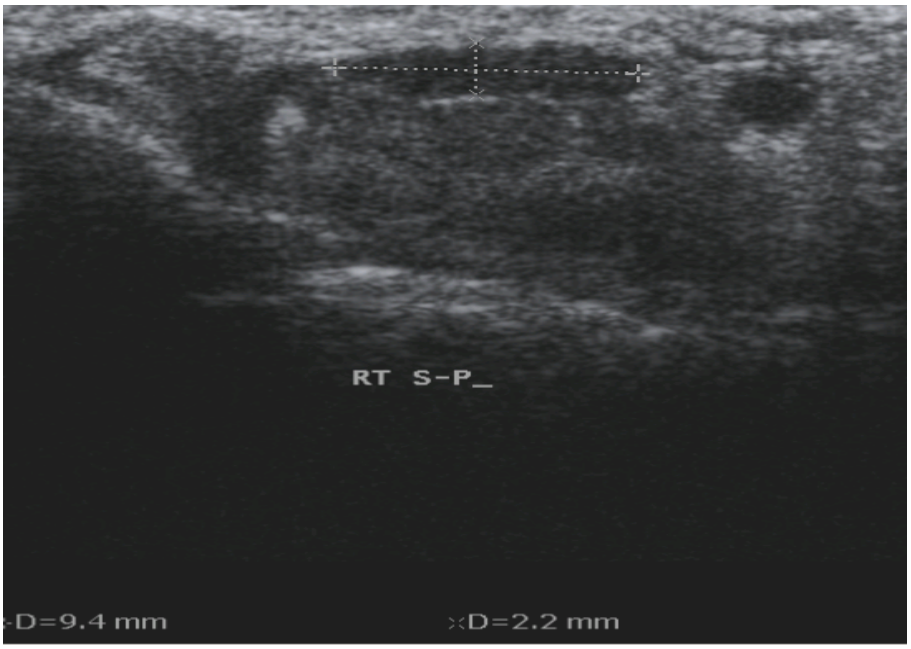
Figure 5

Figure 6 (cross-sectional area at the pisiform level)



The flattening ratio which is defined as the ratio of the major axis of the median nerve to the minor axis was also assessed at the tunnel inlet and outlet (figure 7).

Figure 7 (flattening ratio at the level of pisiform)



Of the 100 subjects included in the study (36 patients with CTS and 64 normal subjects), 18 were found to have unilateral bifid median nerve. The actual numbers of subjects included in the study were 110, but of this number, 10 people were found to have bilateral bifid nerves, a normal variant of the median nerve. Only those with unilateral bifid nerves were included in the study. Among the patient group, there were 8 people with this variant but in the asymptomatic hand. Among the normal, 10 individuals had the variant, hence the wrist with the variant nerve was excluded from the study and the normal wrist was included.

Figure 8:(Bifid median nerve at the level of distal radio-ulnar joint with persistent median artery seen on doppler)

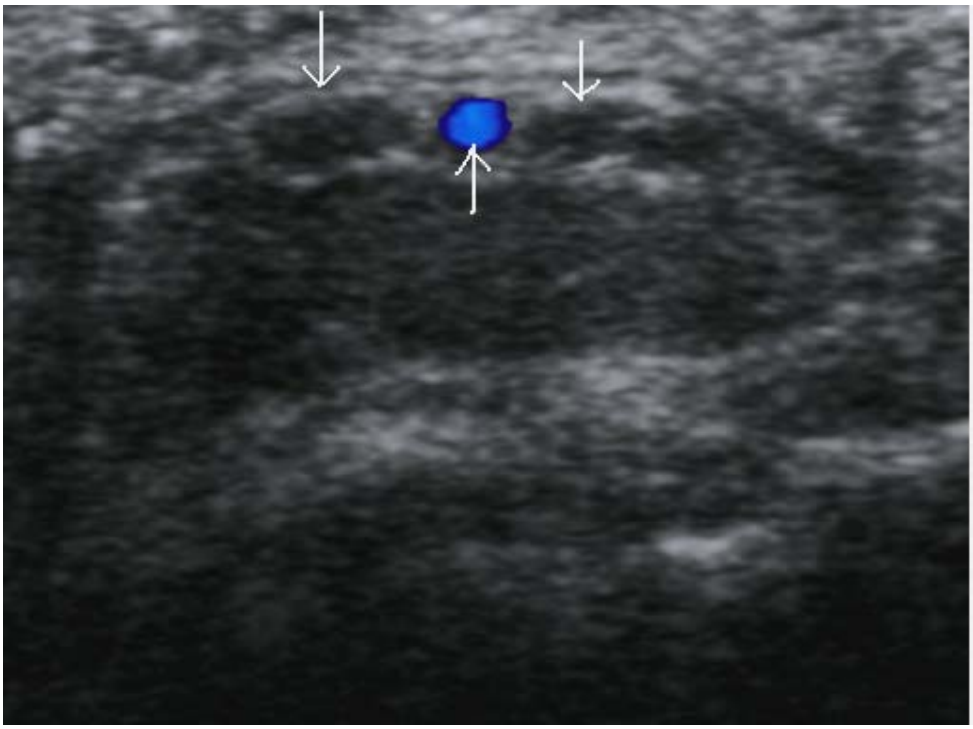
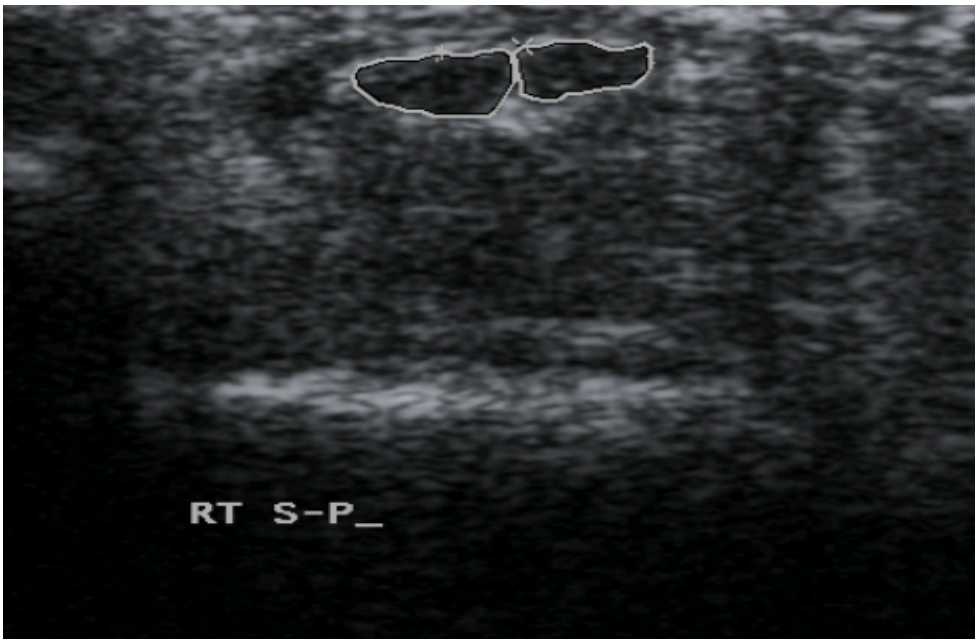


Figure 9: (early bifurcation of the median nerve in the carpal tunnel)



Statistical analysis

The statistical analysis was done using the STATA 8.0 (Statacorp, Texas, USA). The cut off values of the cross-sectional area for each of the three levels (proximal to inlet, at the inlet and at the outlet) were calculated according to the receiver operating characteristic curves (ROC curve) at each level. The co-relation of the positive ultrasound findings at each level and nerve conduction studies was done along with calculation of the positive and negative predictive values. The mean flattening ratio for cases, controls and the combined group were also calculated.

RESULTS

The study was conducted in the Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore. The subjects were patients with symptoms of Carpal Tunnel Syndrome, referred from the out-patient services of the departments of Hand and Leprosy Reconstructive surgery (HLRS), Orthopaedics and PMR, between December 2006 and July 2008 for evaluation with Electrodiagnostic studies for CTS. The patients recruited for the study were those who were found to have carpal tunnel syndrome on NCV/EMG. They were also evaluated for any secondary cause for CTS (diabetes, hypothyroidism, fractures of the wrist). An informed consent was obtained from all the recruited patients.

The total sample size of 100 subjects was required to find a hypothesized sensitivity of 90% with a confidence interval of 85-95%. All statistical analyses were done using STATA 8.0 (STATACORP, TEXAS, USA).

Of the 36 patients recruited for the study 10 were males and 26 were female patients (Figure 10) and among the control population of 64 subjects, there were 33 males and 31 females (Figure 11).

Figure 10: Distribution of males and females among the cases.

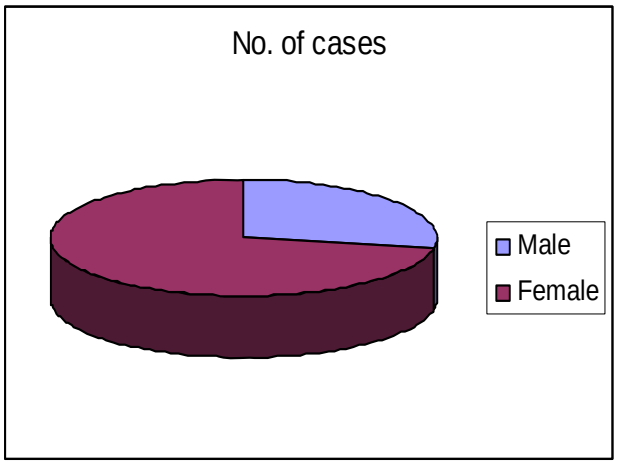
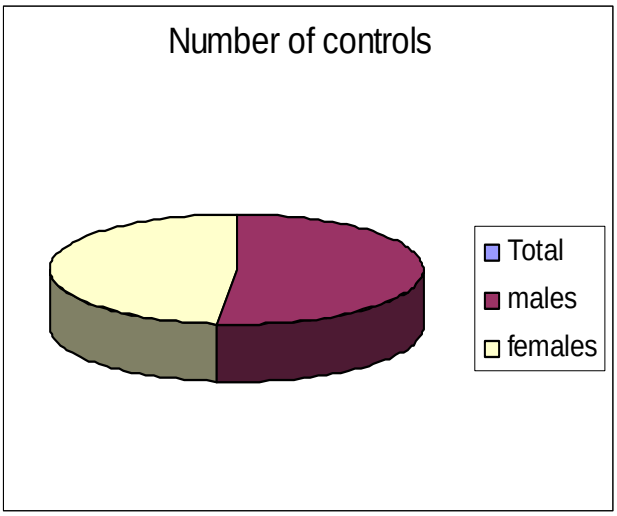
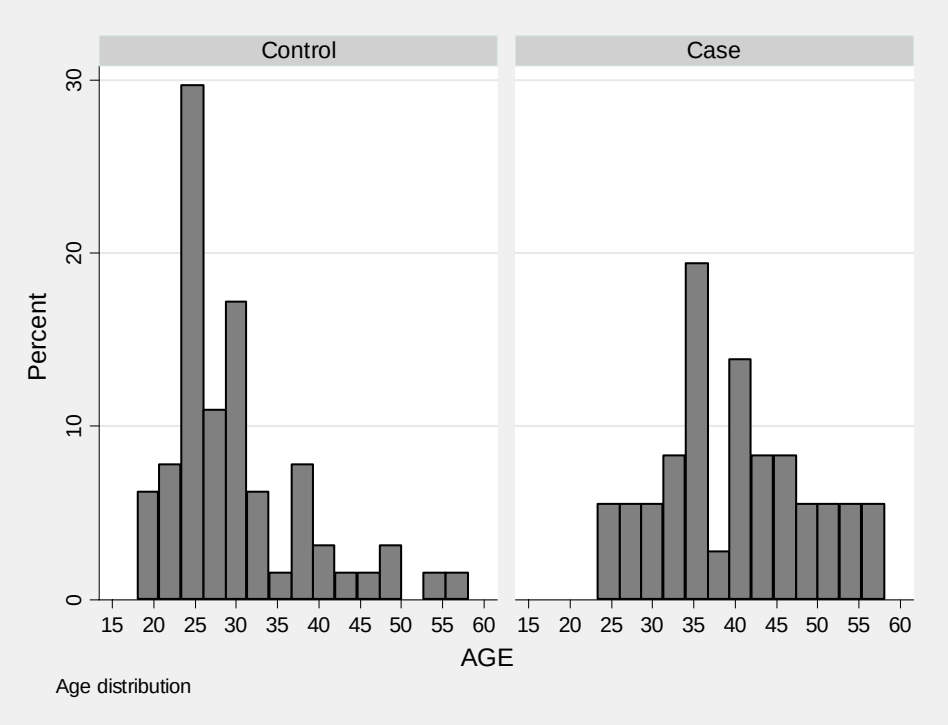


Figure 11: Distribution of males and females among the controls.



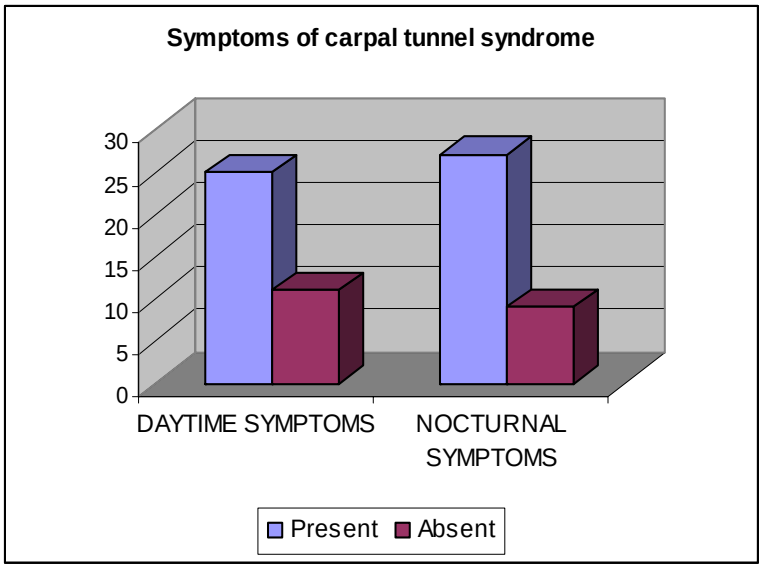
The age distribution in the patient population was between 18-56 years with a mean of 38.33(Figure 12). Among the control group the age distribution was 18-58 years with a mean of 29.89 (Figure 12).

Figure 12: Age distribution among cases and controls.



A detailed history of the symptoms of carpal tunnel syndrome was taken according to the Boston Questionnaire(85) and the patients were categorized as having daytime and/ or nocturnal symptoms. Of the 36 patients, 25 patients had daytime symptoms ranging from mild tingling to hand, wrist and forearm pain to difficulty in carrying out daily chores. 27 patients had nocturnal symptoms ranging from tingling sensation to severe pain in the night causing sleep disturbance (Figure 13).

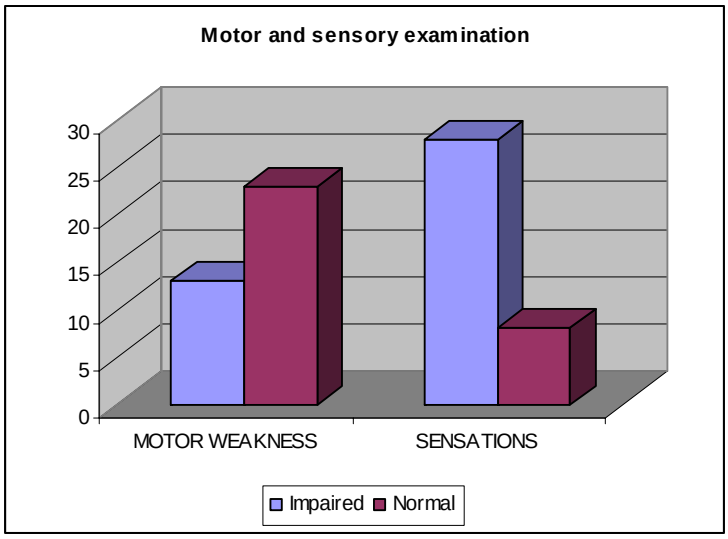
Figure 13: Distribution of daytime and nocturnal symptoms in patients.



A clinical examination was done for any sensory deficits in the median innervated area in the hand (categorized as impaired sensations/ normal sensations) and for motor weakness of the abductor pollicis brevis (categorized as weakness present or normal).

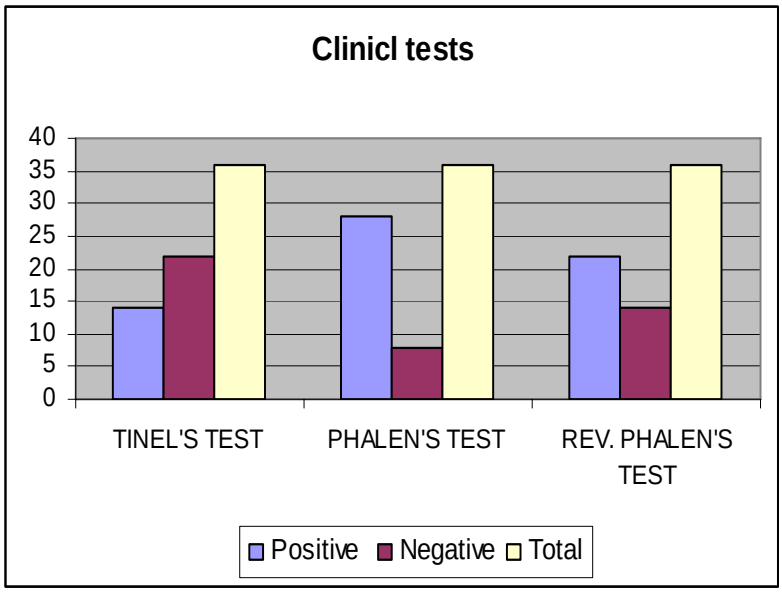
13 out of the 36 patients were found to have motor weakness and 28 people were found to have sensory impairment in the hand in the median innervated areas, namely the thumb, index, middle and radial half of the ring finger. The results are shown in following bar charts (Figure 14).

Figure 14: Distribution of motor and sensory examination among the patients.



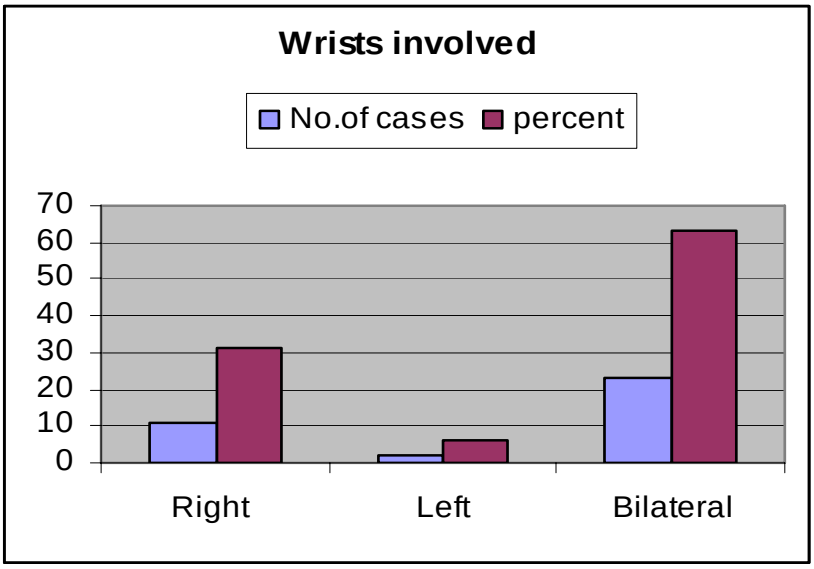
Each patients as well as subjects from the control group were evaluated with the Tinel's sign; Phalen's and reverse Phalen's tests. Of the 36 patients, there were 25 people with daytime symptoms and 27 with nocturnal symptoms (figure 15).

Figure 15: Distribution of clinical tests in the patients group.



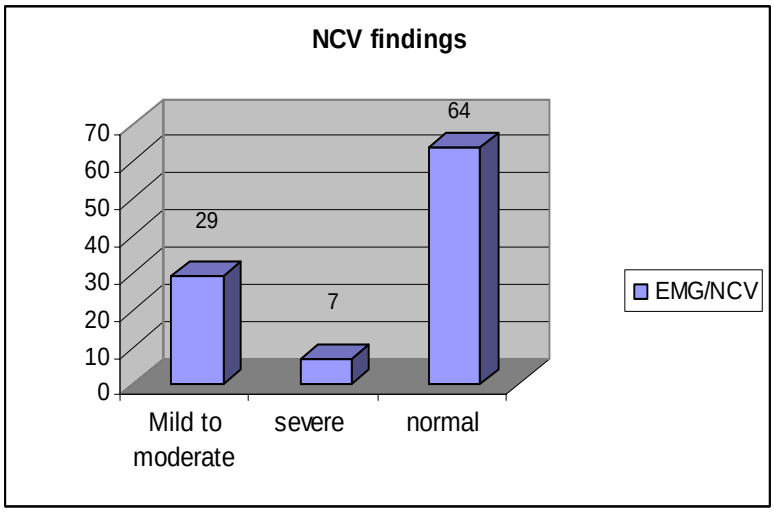
Of the total number of 36 patients, 23 were found to have bilateral carpal tunnel syndrome, 13 were found to have unilateral disease of which 11 patients had right hand disease and 2 patients were found to have left handed disease (figure. 16).

Figure 16: The number of wrists found to have CTS in the patient group.



The 64 subjects of the control group were healthy people recruited from the hospital staff or relatives accompanying the patients, who did not have any signs or symptoms of carpal tunnel syndrome. According to the results of nerve conduction studies were done for all the 100 subjects included in the study they were categorized into 3 groups, 1= mild to moderate CTS, 2= severe CTS and 3= normal. Of the 36 subjects in the patient group, 29 were group 1 with mild to moderate CTS, 7 in group 2 with severe CTS and 64 in the normal group (figure 17).

Figure 17: Distribution of NCV findings in the 100 subjects included in the study.



Following nerve conduction studies, Ultrasonography was done for all the 100 subjects. The ultrasound was performed by two radiologists who were experienced in the field of musculoskeletal Sonography. The measurements of the median nerve at the wrist were taken at 3 levels – at the proximal inlet which corresponded to the distal radio-ulnar joint, the inlet of the carpal tunnel corresponding to the level of the pisiform and at the tunnel outlet corresponding to the level of the hook of hamate. The cross sectional area of the Median nerve at each level was measured by directly tracing with electronic calipers around the margin of the Median nerve. The margin of the median nerve was defined as the margin outside the hypoechoic nerve fasicles and inside the hyperechoic nerve sheath. The flattening ratio, defined as the ratio of major axis to minor axis, was also taken at each level.

18 subjects among the cases as well as the control group were found to have a bifid median nerve, which has been described in literature as a normal variant. Among the 11

patients with right sided carpal tunnel syndrome, one person was found to have a bifid nerve in the left asymptomatic hand. There were 7 patients found to have unilateral bifid nerve among the group having bilateral CTS and of the two patients with left sided CTS, there were none with the variant. In the control group, there were 10 subjects who were found to have unilateral bifid nerve (figure 18). A total of 18 wrists were found to have bifid median nerve at the level of the distal radio-ulnar joint or at the level of inlet to the carpal tunnel.

Figure 18: Percentage of Patients with Bifid median nerve according to the wrists involved.

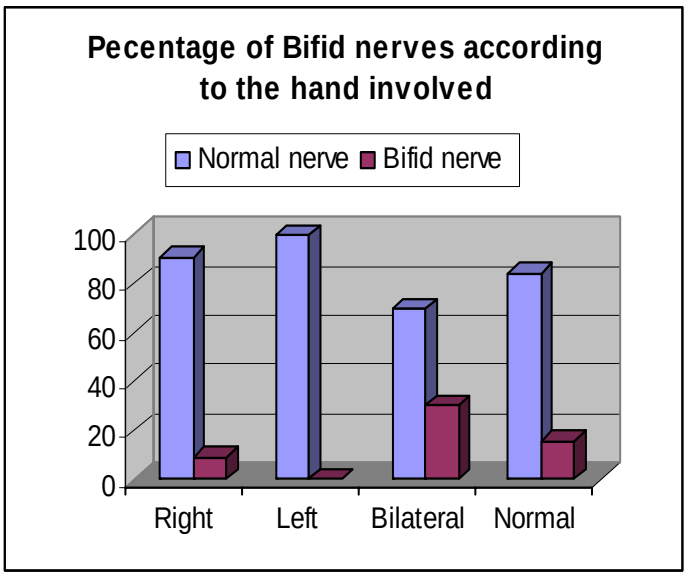
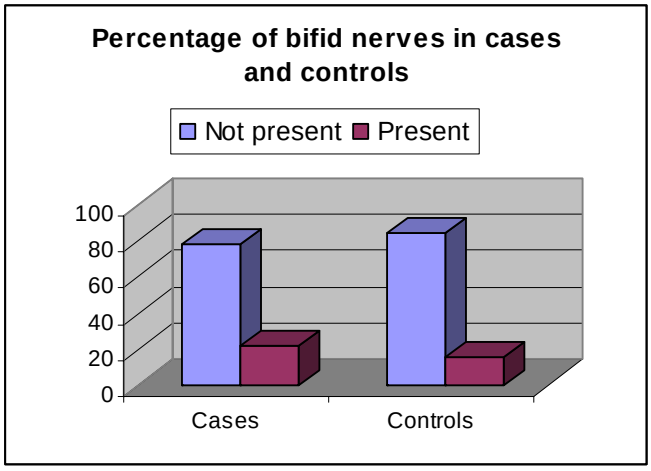


Table 4: Distribution of Bifid nerves among the patient group.

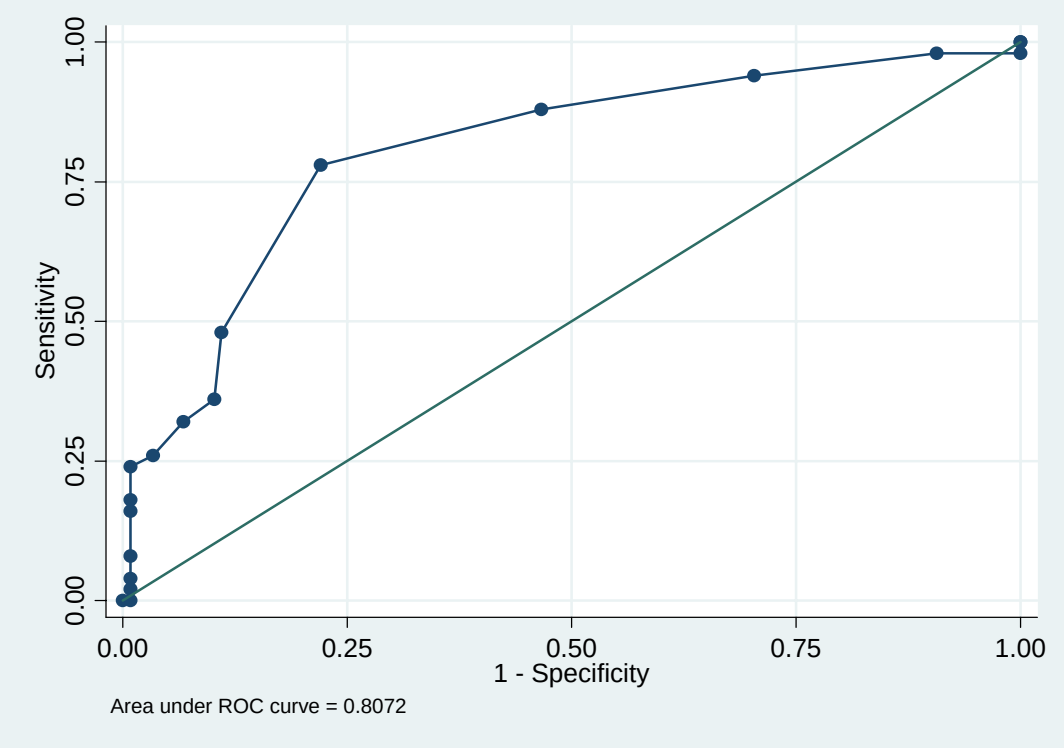
Wrist involved	Case/control group	Wrists with bifid nerve	Total
Right	10	1	11
Left	2	0	2
Bilateral	16	7	23
Normal	54	10	64

Figure 19: Percentage of Bifid Median nerves among the 100 subjects included in the study.

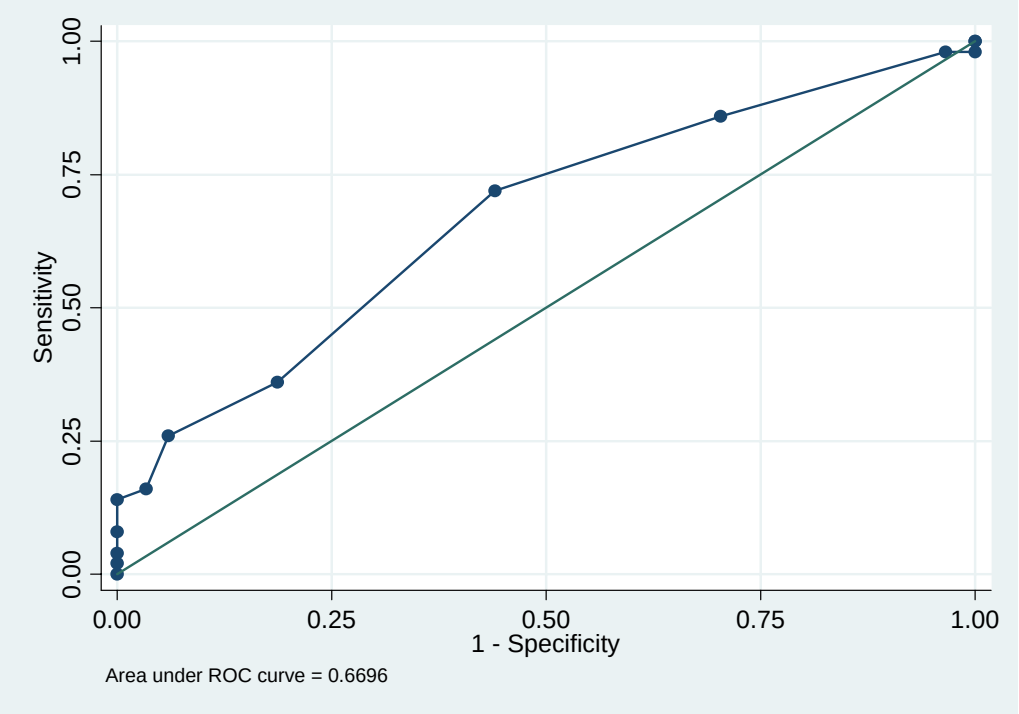


The wrists with the bifid nerve were excluded from the study as two instead of one cross sectional areas of the median nerve would have to be taken at each of the 3 levels on ultrasound. Hence among the case group, 8 wrists were excluded from a total of 59 wrists afflicted with carpal tunnel syndrome. Therefore, a total of 51 wrists of 36 patients were included for the statistical analysis. Among the control group, of the 64 subjects, 10 were found to have unilateral asymptomatic bifid nerves. Hence of the total of 128 wrists, 10 wrists were excluded; therefore 118 wrists of 64 subjects were included for further analysis.

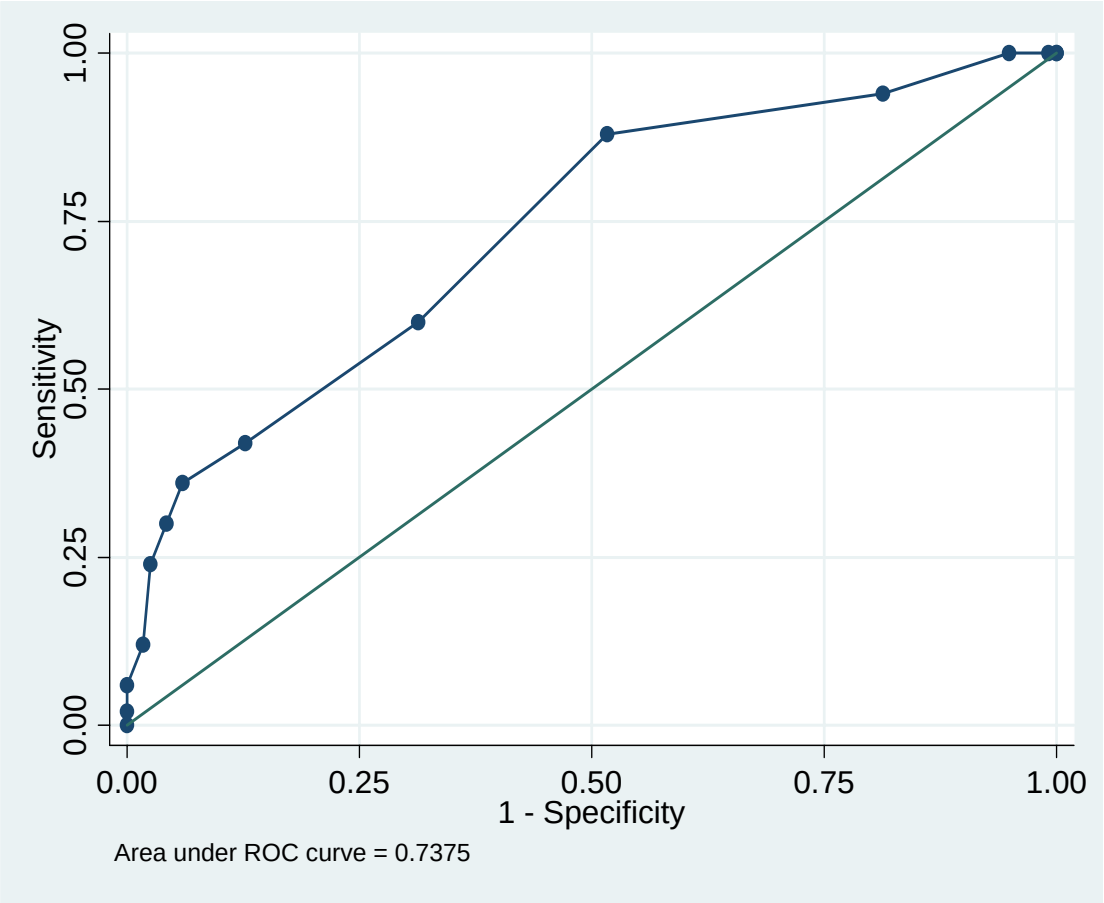
(Figure 20)The ROC curves for the proximal inlet is -



(Figure 21) The ROC curve for the inlet of the carpal tunnel is –



(Figure 22)The ROC curve for the carpal tunnel outlet is –



Multiple values were obtained on analysis of data for the best cut off value. Based on the ROC curve, the values with the best sensitivity and specificity at each level were taken, given in the table below:

Table 5: Sensitivity and specificity for the cut offs of the cross sectional areas of the median nerve at the level proximal to inlet, the inlet and at the outlet.

Level	**Cut off in cm2**	**Sensitivity**	**specificity**
Proximal	>=0.09	68%	68.64%
Inlet	>=0.10	78%	77.97%
Outlet	>=0.08	72%	55.93%

The co-relations between the ultrasound findings and the NCS results were studied at each level i.e. proximal to inlet, at the inlet and at the outlet.

Table 6, **Sensitivity and specificity of the cross sectional area of the median nerve at the distal radio-ulnar joint as compared to EMG (proximal to the inlet of the carpal tunnel).**

Cut off value	**Group 1 (%)**	**Group 2 (%)**	**Group 3 (%)**
< 0.09 cm2	41.46	33.33	68.64
>= 0.09 cm2	58.54	66.67	31.36

The sensitivity of ultrasound to diagnose CTS by the increase in the cross sectional area at the level of the distal radio-ulnar joint for group 1 (mild to moderate CTS on NCS) is 58.54% and that for group 2 (severe CTS) is 66.67%. The specificity of ultrasound compared to the Electrodiagnostic study is 68.64% (table 3).

Table 7, **Sensitivity and specificity of the cross sectional area of the median nerve at the at the inlet of the carpal tunnel** as **compared to EMG.**

Cut off value	**Group 1 (%)**	**Group 2 (%)**	**Group 3 (%)**
< 0.10 cm2	21.95	22.22	92
>= 0.10 cm2	78.05	77.78	22.03

The sensitivity of ultrasound to diagnose CTS by the increase in the cross sectional area at the level of inlet to the carpal tunnel (level of pisiform) in group 1 (mild to moderate CTS on NCS) is 78.05% and that for group 2 (severe CTS) is 77.78%. The specificity of ultrasound compared to the Electrodiagnostic study at this level is 92% (table 4).

Table 8: **Sensitivity and specificity of the cross sectional area of the median nerve at the at the outlet of the carpal tunnel as compared to EMG.**

Cut off value	**Group 1 (%)**	**Group 2 (%)**	**Group 3 (%)**
< 0.08 cm2	29.27	22.22	66
>= 0.08 cm2	70.73	77.78	44.07

The sensitivity of ultrasound to diagnose CTS by the increase in the cross sectional area at the level of the outlet of the carpal tunnel (level of hook of hamate) in group 1 (mild to moderate CTS on NCS) is 70.73% and that for group 2 (severe CTS) is 77.78%. The specificity of ultrasound compared to the Electrodiagnostic study at this level is 66% (table 8).

The sensitivity and specificity for the NCV based group 1 and 2 with an ultrasound positive value at any one level was 90% sensitivity and 45 % specificity (Table 9).

Table 9: Sensitivity and specificity of the combined group of cases and controls when compared to Nerve conduction studies.

	Group 1 and 2 (%)	**Group 3 (%)**
Normal on US	10	45
Positive on US for CTS	90	55

Positive on ultrasound means to say that one value at any of the three levels is above the cut off value for that level to diagnose CTS.

For group 1 (mild to moderate CTS on NCV), the sensitivity is 90.2% having 37 patients with a positive value on US and 4 patients with totally normal Ultrasonography (9.8%). In the group with severe CTS diagnosed on NCV, there were 9 persons totally of whom 8 that is 88.9% having a positive value in ultrasound and 1 person (11.3%) normal on US.

Table 10: Sensitivity of Ultrasound with a positive value at any one of the three levels, for the NCV groups 1 and 2 (found to have CTS).

	Group 1 (mild to moderate CTS)	**Group 2 (severe CTS)**
Normal on US	9.8 %	11.3 %
Positive on US	90.2 %	88.7 %

The flattening ratio for all 100 subjects were taken at the 3 levels and the mean and standard deviated was calculated for the same.

Table 11: Mean of flattening ratio for cases at the level proximal to the inlet, at the inlet and at the outlet.

	No. of wrists	Mean	Standard deviation	Minimum ratio	Maximum ratio
Proximal to inlet	64	2.85	0.64	1.36	4.69
At the inlet	64	2.69	0. 0.62	1.51	4.7
At the outlet	64	3.04	0.72	1. 1.72	5

The mean of flattening ratio for the cases at the level of proximal inlet is 2.85 with standard deviation of 0.63, at the inlet the mean is 2.69 with standard deviation of 0.63 and at the tunnel outlet it is 3.04 with standard deviation of 0.72 (table 11).

Table 12: Mean of flattening ratio for controls at the level proximal to the inlet, at the inlet and at the outlet.

	No. of wrists	Mean	Standard deviation	Minimum ratio	Maximum ratio
Proximal to inlet	118	3.18	0.78	1.79	5.27
At the inlet	118	2.77	0. 0.56	1.48	4.38
At the outlet	118	3.32	0.79	1. 1.11	5.27

The mean of flattening ratio for the controls at the level of proximal inlet is 3.18 with standard deviation of 0.78, at the inlet the mean is 2.77 with standard deviation of 0.56 and at the tunnel outlet it is 3.32 with standard deviation of 0.79 (Table 12).

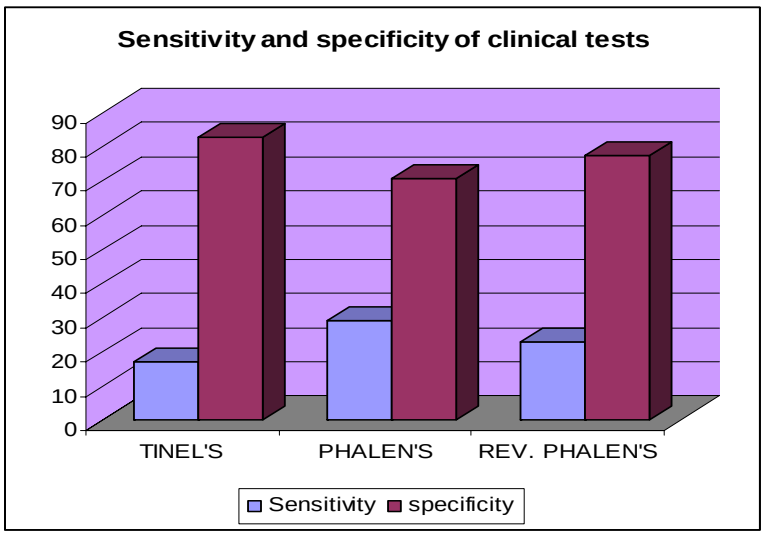
Table 13: mean of flattening ratio for controls and cases, combined at the level proximal to the inlet, at the inlet and at the outlet.

	No. of wrists	Mean	Standard deviation	Minimum ratio	Maximum ratio
Proximal to inlet	182	3.06	0.75	1.36	5.27
At the inlet	182	2.74	0. 0.58	1.48	4.7
At the outlet	182	3.22	0.78	1. 1.11	5.27

The mean of flattening ratio for the combined group at the level of proximal inlet is 3.06 with standard deviation of 0.75, at the inlet the mean is 2.74 with standard deviation of 0.58 and at the tunnel outlet it is 3.22 with standard deviation of 0.78 (Table 11).

A co-relation between the clinical tests and the ultrasound findings were made. A 110 wrists positive at any one of the three levels (proximal to inlet, at the inlet and at the outlet) were taken and the number of wrists among these that were positive for the tests (Tinel's, Phalen's or reverse Phalen's test) were charted. The results are as seen in the bar chart given below (figure 23):

Figure 23: Sensitivity and specificity of clinical tests as compared to US results.



All the three tests were found to have a good specificity - 82.73% at the level proximal to the inlet, 70.91 % at the inlet and 77.27 at the outlet. However, the sensitivity of the tests to the ultrasound findings is poor – 17.27 at the level proximal to the inlet, 29.09 at the level of the inlet and 22.73 at the outlet.

Also the positive predictive value and the negative predictive values of ultrasound vs. nerve conduction studies were calculated at each level (Figure 24).

1. Proximal inlet

Positive predictive value = 45

Negative predictive value = 80

2. Inlet

Positive predictive value = 60

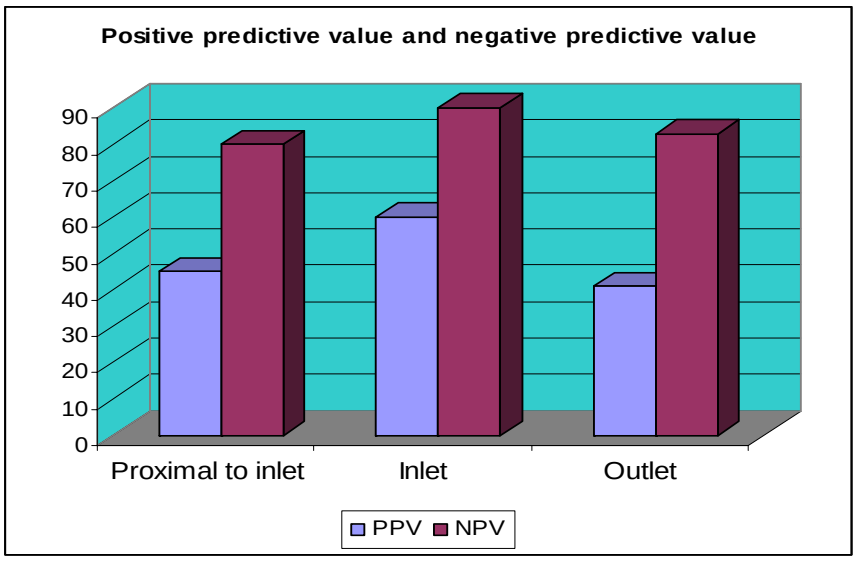
Negative predictive value = 90

3. Outlet

Positive predictive value = 41

Negative predictive value = 83

Figure 24: The positive and negative predictive values at the level proximal to the inlet, at the inlet and at the outlet.



During the course of study (2 years), there were 10 subjects among the 36 patients who underwent surgery (open carpal tunnel release) for CTS (figure 25). Of the 10 patients who underwent the surgery, there were 9 of them who were diagnosed to have bilateral carpal tunnel syndrome (figure 26).

Figure 25: Number of patients operated

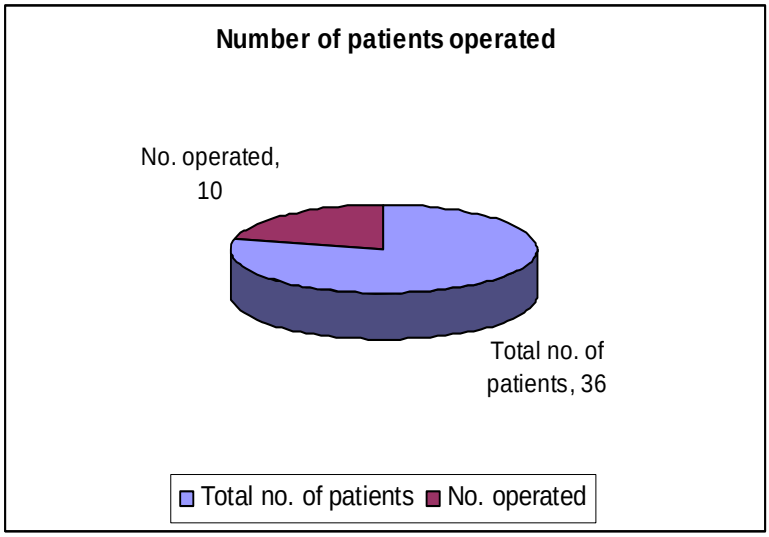
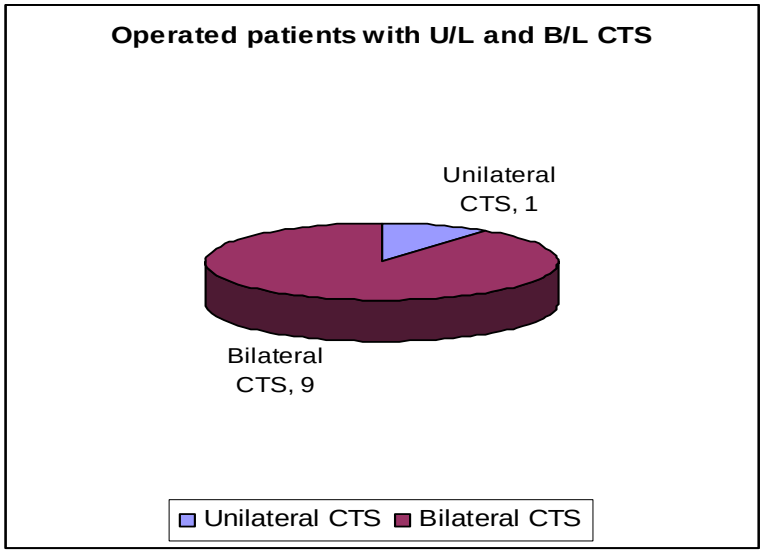
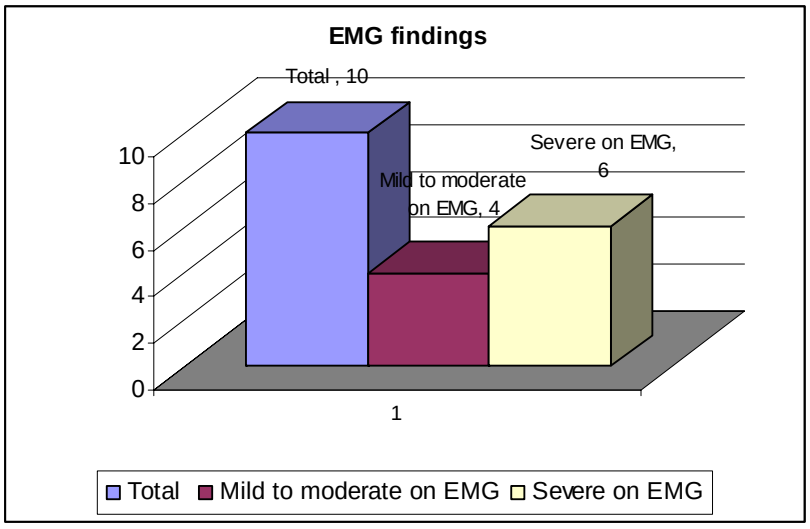


Figure 26: Number of operated patients with unilateral/ bilateral wrists.



6 of the 10 patients, who underwent surgery, were found to have severe CTS on nerve conduction studies and 4 were in the mild to moderate category of nerve conduction study (figure 27).

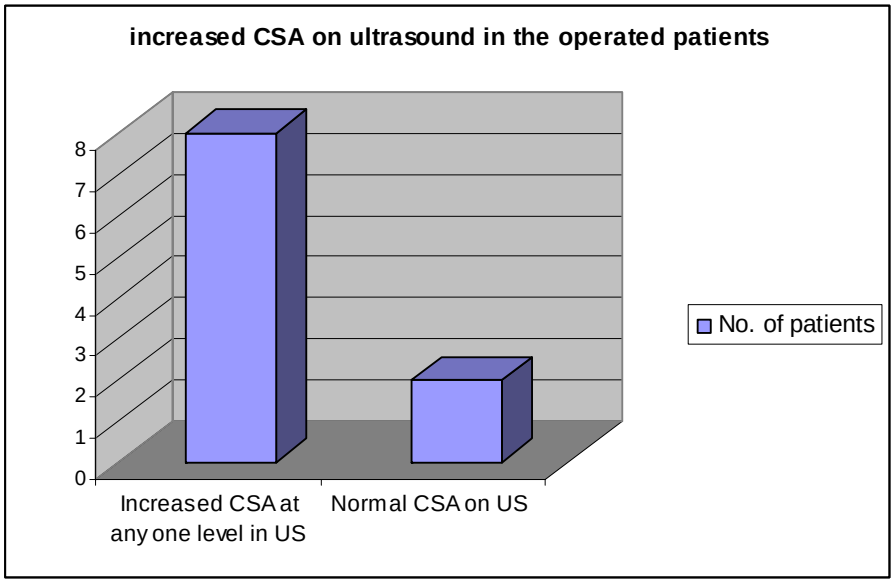
Figure 27: Number of patients in the 3 NCV categories who underwent surgical management.



The cross sectional area of the median nerve at any one level, that is at the distal radio-ulnar joint, the inlet of the tunnel or at the outlet of the tunnel, was found to be increased in 8 of the 10 patients who underwent surgery. The 2 remaining patients had normal cross sectional area of the median nerve at all the three levels.

Of the 9 patients who were found to have bilateral CTS on nerve conduction studies, 4 subjects underwent surgery for both the wrists. The wrist that was found to have more severe nerve conduction abnormalities was operated first and followed by the less severe wrist within a period of 6-8 months.

Figure 28: Number of patients who underwent surgical management, found positive at any one level in US.



Among the 9 patients with bilateral CTS, 2 subjects had bifid median nerve. One patient underwent surgical release of the transverse ligament of the wrist with the normal variant, while the second patient though was found to have CTS on NCV in both wrists, but the severity of the disease was more on the side without the bifid nerve.

DISCUSSION

The diagnosis of carpal tunnel syndrome usually relies on typical signs, symptoms and outcomes of Electrodiagnostic studies. Single signs and symptoms have shown to have limited diagnostic accuracy, while Electrodiagnostic methods are time consuming, expensive and not widely available. Therefore there has been a search for an alternative modality to confirm the diagnosis of CTS. Ultrasonographic detection of pathologic swelling of the median nerve by assessing its cross sectional area has been in the recent times found to be most convenient and least expensive.

A PubMed search identified several publications containing information on use of Ultrasonography in the diagnosis of CTS; detailed findings are summarized in Table 9 (annexure). The table shows a very varied picture of inclusion criteria of CTS cases, Ultrasonography, NCV, statistical methods, and outcome results, making a comparison difficult. Ultrasonography is useful in CTS diagnosis, providing anatomic images of the median nerve, neighbouring structures, and mass-occupying space in the carpal canal. The advantages of Ultrasonography is that it is low cost, takes short duration to perform the investigation compared to nerve conduction studies and commonly available, besides it is painless and non-invasive; and gives dynamic images. US is operator dependent, but shows high reproducibility after adequate training of the operators (167).

The US measurement used in CTS diagnosis is the cross sectional area of the nerve at various levels of the carpal canal, the flattening ratio, the swelling ratio, and the increased palmer bowing of the flexor retinaculum. In some studies cross sectional area was performed at a single level (17, 18, 21, 117, 118, 123, 168), mostly at the proximal carpal tunnel. In several studies cross sectional area was measured by ellipsoid formula(17) (24, 28, 31, 116, 117), but a more accurate measure is obtained

by using continuous boundary trace of the nerve, because the nerve does not always have a perfect ellipsoid shape . However, some studies demonstrated that similar results are obtained by both methods (17, 18, 35, 118). The sensitivity and specificity of Ultrasonography measures vary widely among studies. Many authors demonstrated that the increase in cross sectional area at the tunnel inlet had the highest sensitivity and specificity (17, 19, 21, 117, 122, 123); moreover, the measurement at this level was easier to perform. In this study also the cross sectional area at inlet of the carpal tunnel showed higher sensitivity and specificity (78% and 77.97%) for CTS as compared to the proximal inlet and outlet of the tunnel.

Among the various studies available, there was also disagreement about the exact localization of tunnel inlet. Most authors considered the proximal edge of the flexor retinaculum, approximately at the level of the distal radio-ulnar joint, as the tunnel inlet, while others considered the pisiform bone as the landmarks (169). In this study, we have taken the distal radio-ulnar joint as the level proximal to the tunnel, the level of the pisiform as the level of the inlet and the level of the hook of the hamate as the outlet of the carpal tunnel.

Studies have shown that ultrasound measurements have a good inter- and intra-observer reliability.13

Nakamichi and Tachibana directly compared the measurements of the median nerve obtained sonographically with the measurements found in anatomical cross-sections in cadaver limbs.(26). Ultrasound is a precise method for determining these measurements. This was later confirmed by Kamolz et al(170).

The sensitivity of the cross sectional areas ranged from 48% to 89% (17-19, 21, 30, 116, 119, 121-124, 171, 172) and the CSA cut off at which the value was considered abnormal varied from 9 mm2 (17, 19) to 15 mm2 (28). In our study, the cut off for an

abnormal nerve was taken as 0.09 cm2 at the level proximal to the inlet, 0.10 cm2 at the inlet and 0.08 cm2 at the tunnel outlet with sensitivities of 68%, 78% and 72% respectively and specificities of 68.64%, 77.97% and 55.93%.

Sensitivities of increased palmer bowing of the flexor retinaculum varied from 40% to 81% (24, 31, 116, 117, 173), and sensitivities of flattening ratio ranged from 37% to 100% (17, 18, 31, 117, 125).

In our study, the sensitivity and specificity of the flattening ratio was not calculated as it did not show any significant co-relation with the presence of the disease. However, the mean of the flattening ratios for the cases and the controls was taken separately (Table 9,10,11).

The discrepancies in the varied range of outcome result from many factors: selection criteria of patients and controls, gold standard for diagnosis of CTS, Electrodiagnostic methods, levels of CSA measurement, and US cut off values. A bias of the results may also be due to incorrect selection of the control group. An instance of selection leading to a bias - sometimes the asymptomatic wrist of CTS cases was included in the control group (18, 24). In almost all studies the gold standard of CTS diagnosis was based on clinical and abnormal Electrodiagnostic tests, and sometimes the most sensitive tests, such as short segment study or comparative test of median-ulnar distal sensory latency, were not performed.

In contrast, only a few studies used clinical findings only as the gold standard (118, 119, 123, 168, 173). Only this type of study is able to compare US specificity and sensitivity with those of the Electrodiagnostic tests. The few literature data reported different results on NCV specificity (26, 119, 123, 124). In particular, Swen et al (123) reported a very low NCV specificity (19%). They used as the gold standard patients with 90% relief of symptoms after surgery (123) and received many criticisms

on their selection criteria of CTS cases. In the literature, only the study by Altinok et al (173) took into account mild-moderate cases. These authors defined mild cases as wrists with normal NCV and moderate cases as wrists with abnormal NCV, and demonstrated that abnormal US findings were present in 30% of 20 mild cases and in 100% of 20 moderate cases (173). Moreover, Koyuncuoglu et al (118) studied 59 wrists with negative Electrodiagnostic tests and showed that CSA-I was abnormal (10.5 mm2) in 30.5% of wrists with clinically diagnosed CTS. In accordance with AAEM Electrodiagnostic protocol, when standard methods did not show any conduction anomalies of the median nerve, comparative tests (ulnar/ median distal SCV comparison) or short segment conduction velocity was used. These tests have high sensitivity and high specificity (105). However, some authors consider NCV an "unnecessary luxury" and for others NCV causes discomfort and is considered expensive and time consuming (21). We disagree that NCV is time consuming and uncomfortable, because an experienced electromyographer can perform NCV for CTS according to AAEM protocol in 20 minutes, using surface electrodes and small current intensity. Needle electromyography is rarely necessary. Thus laboratories can use their own normal reference values, making this scale a valuable tool for comparing electrophysiologic results from different laboratories that use different techniques and reference values. This scale was also used by other authors in US studies (32, 119). In our study, we have used our normal reference standards based on the controls done in the lab and also the guidelines by Kimura (1986) and Kalita and Misra (15).

CONCLUSION

1. The cut offs of the cross sectional areas of the median nerve at the three anatomical levels on Ultrasonography were taken at the best sensitivity and specificity according to the ROC curve –
   - At the distal radio-ulnar joint (at the level proximal to the carpal tunnel inlet) is 68% sensitivity and 68% specificity.
   - At the level of carpal tunnel inlet (at the pisiform level) 78% sensitivity and 77% specificity.
   - At the level of the carpal tunnel outlet (level of the hook of hamate) the sensitivity is 72% and specificity is 56%

   Based on the above sensitivity and specificity values, the best level to look for the compression of the nerve by increase in the cross sectional area is at the level of the carpal tunnel inlet.
2. In this study, the sensitivity of ultrasound to detect carpal tunnel syndrome by the increase in the cross sectional area of the median nerve as compared to the nerve conduction studies is 90% with the US value may be positive at any one anatomical level. The specificity for this is 45%. Hence Ultrasonography is a good screening investigation.
3. Ultrasonography has been found to be more widely available as compared to nerve conduction studies. It is also less time consuming (average time 15 minutes) and cost effective as compared to the nerve conduction studies. Along with this , Ultrasonography has also the advantage of giving information about the morphology of the nerve and its surrounding structures. Therefore, Ultrasonography can be used as an alternative diagnostic investigation for Carpal tunnel syndrome.

RECOMMENDATIONS FOR FUTURE STUDIES

The study was done to see the cross sectional area of the median nerve at the time of presentation with the symptoms and signs of carpal tunnel syndrome. The nerve at this point of time would be oedematous due to compression. There were 10 patients among the 36 from the patient group who underwent open carpal tunnel release surgery. Eight of the ten patients who underwent surgery were found to have an increase in the cross sectional area on Ultrasonography at any one of the three levels. The remaining 26 patients underwent conservative management in the form of splinting and/ or local steroid injection. Following treatment, the nerve compression decreases which leads to improvement in the blood supply and axonal transport of the nerve thereby decrease in oedema and hence decrease in the cross sectional area with improvement in the conductions.

In this study we have not been able to look at the morphology and the cross sectional area of the median nerve following conservative/surgical management.

For further studies, it may be useful to look for the cross sectional area of the median nerve at the 3 different levels (at the distal radio-ulnar joint, the level of the pisiform and at the level of the hamate) 3-6 months post surgery or conservative management along with concomitant nerve conduction studies and compare with the pre-operative values. This may be especially useful in those patients who continue to have symptoms despite treatment especially surgical management.

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APPENDIX - 1

INFORMED CONSENT FORM

Title of the study: Ultrasonography – an alternative diagnostic tool for Carpal Tunnel Syndrome.

Institution: Department of Physical Medicine & Rehabilitation, CMC, Vellore.

Nature & Purpose of Study: You are being requested to participate in a Research Study to find the sensitivity and specificity of Ultrasonography as a diagnostic tool for diagnosis of Carpal Tunnel Syndrome in order to use it as an alternative investigational modality.

Expected duration of involvement: During your stay as an out patient for investigations.

Confidentiality: your personal data collected will be processed only for the Research purpose in relation to the study. Your participation will in no way affect the treatment of your condition.

Consent: The above information has been explained to me in the language best understood by me before signing this consent form.

Signature of Patient

Date:

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

PATIENT IDENTITY	AGE	SEX	HAND	RIGHT /LEFT	DAYTIME SYMPTOMS	NOCTURNAL SYMPTOMS	MOTOR WEAKNESS	SENSATIONS	TINEL'S TEST	PHALEN'S TEST	REV. PHALEN'S TEST
237228D	38	2	3	2	1	1	1	2	2	2	2
177155D	40	2	1	0	1	1	2	2	2	2	2
075924D	41	2	3	1	1	1	1	1	2	1	1
055489D	35	2	3	3	1	1	2	2	2	1	2
104678D	35	2	2	0	1	1	1	1	1	1	1
868951	33	1	3	1	1	1	2	2	2	1	1
166654D	36	2	3	1	1	1	1	2	1	1	1
275413D	40	2	1	0	1	2	1	1	1	1	2
193453D	35	1	3	3	1	2	2	1	2	1	2
506448C	56	2	3	1	1	1	1	2	1	1	1
093875B	34	2	3	3	2	1	2	2	2	1	1
325682C	55	2	3	1	2	1	2	2	1	1	1
185246D	53	2	3	1	1	1	1	2	2	2	2
152017D	40	2	1	0	1	1	2	1	1	1	1
078485B	56	1	1	0	2	1	2	2	2	1	1
108591C	43	1	3	2	1	1	2	2	1	1	1
019738D	25	2	3	1	2	1	1	2	2	1	2
553707C	45	2	1	0	1	1	1	2	1	1	1
221591D	44	2	2	0	2	1	2	2	2	2	2
164672D	47	2	3	1	1	1	1	2	2	1	2
192446D	50	2	3	3	1	2	2	2	2	2	2
573938B	36	2	3	2	1	2	2	2	2	1	1
092861D	35	2	3	3	1	1	2	2	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

020478D	31	1	3	1	1	1	1	2	1	1	1
042923D	36	2	3	1	1	2	2	2	2	1	1
041400D	47	2	1	0	2	2	2	2	1	1	1
078333D	50	1	3	1	1	1	1	2	1	1	1
215559D	28	2	1	0	1	1	2	2	2	1	1
081396D	51	2	3	3	1	1	2	2	2	2	2
120153D	31	1	1	0	2	2	2	1	2	1	1
042894D	43	1	3	3	1	1	2	2	1	1	2
095386D	52	1	3	1	2	1	1	2	1	1	1
954835C	28	1	3	3	2	2	2	1	2	1	1
190260D	34	2	1	0	2	1	2	2	1	1	1
003760d	40	2	1	0	1	1	2	2	2	1	1
050847D	26	2	1	0	2	2	2	1	2	2	2
114437D	23.7	1	4	0	2	2	2	1	2	2	2
058082A	30.3	1	4	0	2	2	2	1	2	2	2
076985D	26	2	4	0	2	2	2	1	2	2	2
232945D	26	2	4	0	2	2	2	1	2	2	2
378854C	29.4	1	4	0	2	2	2	1	2	2	2
293723B	43	1	4	0	2	2	2	1	2	2	2
121872D	23.2	2	4	0	2	2	2	1	2	2	2
469605C	24.1	2	4	0	2	2	2	1	2	2	2
677310C	35	2	4	0	2	2	2	1	2	2	2
275082D	24.2	2	4	0	2	2	2	1	2	2	2
111987D	24.1	2	4	0	2	2	2	1	2	2	2
167632A	28.4	1	4	0	2	2	2	1	2	2	2
777192B	27	1	4	0	2	2	2	1	2	2	2
903869C	25	1	4	0	2	2	2	1	2	2	2
192987D	26	1	4	0	2	2	2	1	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

591715A	24	1	4	0	2	2	2	1	2	2	2
882138C	19.3	1	4	0	2	2	2	1	2	2	2
103999B	21.5	1	4	0	2	2	2	1	2	2	2
790550C	29	1	4	0	2	2	2	1	2	2	2
718720C	29	1	4	0	2	2	2	1	2	2	2
047675C	24.1	2	4	0	2	2	2	1	2	2	2
410225C	24	2	4	0	2	2	2	1	2	2	2
114745D	25	2	4	0	2	2	2	1	2	2	2
582809C	33	2	4	0	2	2	2	1	2	2	2
865596C	21.7	1	4	0	2	2	2	1	2	2	2
193586D	25	1	4	0	2	2	2	1	2	2	2
089083D	18.3	1	4	0	2	2	2	1	2	2	2
644510B	27.1	2	4	0	2	2	2	1	2	2	2
908001B	26	1	4	0	2	2	2	1	2	2	2
217315D	25	2	4	0	2	2	2	1	2	2	2
780402C	27.1	2	4	0	2	2	2	1	2	2	2
262607D	30	1	4	0	2	2	2	1	2	2	2
087170C	22	1	4	0	2	2	2	1	2	2	2
502907C	25.4	2	4	0	2	2	2	1	2	2	2
243498D	39.1	1	4	0	2	2	2	1	2	2	2
262669D	46	1	4	0	2	2	2	1	2	2	2
232124D	42	2	4	0	2	2	2	1	2	2	2
213533D	38	2	4	0	2	2	2	1	2	2	2
109076D	19	2	4	0	2	2	2	1	2	2	2
217031D	48	2	4	0	2	2	2	1	2	2	2
128551D	27	2	4	0	2	2	2	1	2	2	2
412971C	31	2	4	0	2	2	2	1	2	2	2
190785D	37	2	4	0	2	2	2	1	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

524577B	34	2	4	0	2	2	2	1	2	2	2
181785D	31	2	4	0	2	2	2	1	2	2	2
110964D	33	2	4	0	2	2	2	1	2	2	2
141275D	33	1	4	0	2	2	2	1	2	2	2
980145A	38	2	4	0	2	2	2	1	2	2	2
315350C	42	1	4	0	2	2	2	1	2	2	2
105897D	29	2	4	0	2	2	2	1	2	2	2
755823C	26	2	4	0	2	2	2	1	2	2	2
496011	58	1	4	0	2	2	2	1	2	2	2
112698D	26	1	4	0	2	2	2	1	2	2	2
142444D	23	1	4	0	2		2	1	2	2	2
864968C	24	1	4	0	2	2	2	1	2	2	2
232062D	29.8	2	4	0	2	2	2	1	2	2	2
369624C	29	1	4	0	2	2	2	1	2	2	2
115521D	39	1	4	0	2	2	2	1	2	2	2
110373A	29.5	1	4	0	2	2	2	1	2	2	2
342144C	27.1	2	4	0	2	2	2	1	2	2	2
136183A	28.1	2	4	0	2	2	2	1	2	2	2
141518B	50	1	4	0	2	2	2	1	2	2	2
257981D	53	1	4	0	2	2	2	1	2	2	2
073414D	18	1	4	0	2	2	2	1	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

PATIENT IDENTITY	AGE	SEX	HAND	RIGHT /LEFT	DAYTIME SYMPTOMS	NOCTURNAL SYMPTOMS	MOTOR WEAKNESS	SENSATIONS	TINEL'S TEST	PHALEN'S TEST	REV. PHALEN'S TEST
237228D	38	2	3	2	1	1	1	2	2	2	2
177155D	40	2	1	0	1	1	2	2	2	2	2
075924D	41	2	3	1	1	1	1	1	2	1	1
055489D	35	2	3	3	1	1	2	2	2	1	2
104678D	35	2	2	0	1	1	1	1	1	1	1
868951	33	1	3	1	1	1	2	2	2	1	1
166654D	36	2	3	1	1	1	1	2	1	1	1
275413D	40	2	1	0	1	2	1	1	1	1	2
193453D	35	1	3	3	1	2	2	1	2	1	2
506448C	56	2	3	1	1	1	1	2	1	1	1
093875B	34	2	3	3	2	1	2	2	2	1	1
325682C	55	2	3	1	2	1	2	2	1	1	1
185246D	53	2	3	1	1	1	1	2	2	2	2
152017D	40	2	1	0	1	1	2	1	1	1	1
078485B	56	1	1	0	2	1	2	2	2	1	1
108591C	43	1	3	2	1	1	2	2	1	1	1
019738D	25	2	3	1	2	1	1	2	2	1	2
553707C	45	2	1	0	1	1	1	2	1	1	1
221591D	44	2	2	0	2	1	2	2	2	2	2
164672D	47	2	3	1	1	1	1	2	2	1	2
192446D	50	2	3	3	1	2	2	2	2	2	2
573938B	36	2	3	2	1	2	2	2	2	1	1
092861D	35	2	3	3	1	1	2	2	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

020478D	31	1	3	1	1	1	1	2	1	1	1
042923D	36	2	3	1	1	2	2	2	2	1	1
041400D	47	2	1	0	2	2	2	2	1	1	1
078333D	50	1	3	1	1	1	1	2	1	1	1
215559D	28	2	1	0	1	1	2	2	2	1	1
081396D	51	2	3	3	1	1	2	2	2	2	2
120153D	31	1	1	0	2	2	2	1	2	1	1
042894D	43	1	3	3	1	1	2	2	1	1	2
095386D	52	1	3	1	2	1	1	2	1	1	1
954835C	28	1	3	3	2	2	2	1	2	1	1
190260D	34	2	1	0	2	1	2	2	1	1	1
003760d	40	2	1	0	1	1	2	2	2	1	1
050847D	26	2	1	0	2	2	2	1	2	2	2
114437D	23.7	1	4	0	2	2	2	1	2	2	2
058082A	30.3	1	4	0	2	2	2	1	2	2	2
076985D	26	2	4	0	2	2	2	1	2	2	2
232945D	26	2	4	0	2	2	2	1	2	2	2
378854C	29.4	1	4	0	2	2	2	1	2	2	2
293723B	43	1	4	0	2	2	2	1	2	2	2
121872D	23.2	2	4	0	2	2	2	1	2	2	2
469605C	24.1	2	4	0	2	2	2	1	2	2	2
677310C	35	2	4	0	2	2	2	1	2	2	2
275082D	24.2	2	4	0	2	2	2	1	2	2	2
111987D	24.1	2	4	0	2	2	2	1	2	2	2
167632A	28.4	1	4	0	2	2	2	1	2	2	2
777192B	27	1	4	0	2	2	2	1	2	2	2
903869C	25	1	4	0	2	2	2	1	2	2	2
192987D	26	1	4	0	2	2	2	1	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

591715A	24	1	4	0	2	2	2	1	2	2	2
882138C	19.3	1	4	0	2	2	2	1	2	2	2
103999B	21.5	1	4	0	2	2	2	1	2	2	2
790550C	29	1	4	0	2	2	2	1	2	2	2
718720C	29	1	4	0	2	2	2	1	2	2	2
047675C	24.1	2	4	0	2	2	2	1	2	2	2
410225C	24	2	4	0	2	2	2	1	2	2	2
114745D	25	2	4	0	2	2	2	1	2	2	2
582809C	33	2	4	0	2	2	2	1	2	2	2
865596C	21.7	1	4	0	2	2	2	1	2	2	2
193586D	25	1	4	0	2	2	2	1	2	2	2
089083D	18.3	1	4	0	2	2	2	1	2	2	2
644510B	27.1	2	4	0	2	2	2	1	2	2	2
908001B	26	1	4	0	2	2	2	1	2	2	2
217315D	25	2	4	0	2	2	2	1	2	2	2
780402C	27.1	2	4	0	2	2	2	1	2	2	2
262607D	30	1	4	0	2	2	2	1	2	2	2
087170C	22	1	4	0	2	2	2	1	2	2	2
502907C	25.4	2	4	0	2	2	2	1	2	2	2
243498D	39.1	1	4	0	2	2	2	1	2	2	2
262669D	46	1	4	0	2	2	2	1	2	2	2
232124D	42	2	4	0	2	2	2	1	2	2	2
213533D	38	2	4	0	2	2	2	1	2	2	2
109076D	19	2	4	0	2	2	2	1	2	2	2
217031D	48	2	4	0	2	2	2	1	2	2	2
128551D	27	2	4	0	2	2	2	1	2	2	2
412971C	31	2	4	0	2	2	2	1	2	2	2
190785D	37	2	4	0	2	2	2	1	2	2	2

APPENDIX-2
DATA FROM HISTORY AND EXAMINATION

524577B	34	2	4	0	2	2	2	1	2	2	2
181785D	31	2	4	0	2	2	2	1	2	2	2
110964D	33	2	4	0	2	2	2	1	2	2	2
141275D	33	1	4	0	2	2	2	1	2	2	2
980145A	38	2	4	0	2	2	2	1	2	2	2
315350C	42	1	4	0	2	2	2	1	2	2	2
105897D	29	2	4	0	2	2	2	1	2	2	2
755823C	26	2	4	0	2	2	2	1	2	2	2
496011	58	1	4	0	2	2	2	1	2	2	2
112698D	26	1	4	0	2	2	2	1	2	2	2
142444D	23	1	4	0	2		2	1	2	2	2
864968C	24	1	4	0	2	2	2	1	2	2	2
232062D	29.8	2	4	0	2	2	2	1	2	2	2
369624C	29	1	4	0	2	2	2	1	2	2	2
115521D	39	1	4	0	2	2	2	1	2	2	2
110373A	29.5	1	4	0	2	2	2	1	2	2	2
342144C	27.1	2	4	0	2	2	2	1	2	2	2
136183A	28.1	2	4	0	2	2	2	1	2	2	2
141518B	50	1	4	0	2	2	2	1	2	2	2
257981D	53	1	4	0	2	2	2	1	2	2	2
073414D	18	1	4	0	2	2	2	1	2	2	2